



INTRODUCTION TO  
DISEASES OF THE CHEST



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BY

JAMES MAXWELL

M.D. (Lond.), F.R.C.P. (Lond.)

PHYSICIAN, ROYAL CHEST HOSPITAL; PHYSICIAN TO THE MINISTRY'S MARY K HAY UNIT  
CONSULTING PHYSICIAN, ROYAL NATIONAL SANATORIUM, Bournemouth  
LATE PHYSICIAN, ST BARTHOLOMEW'S HOSPITAL

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## PREFACE TO THE THIRD EDITION

INCREASING experience in the recognition of early disease in the respiratory tract leads to the conclusion that physical examination by itself is not enough. It is never possible to be sure that the bronchi and lungs are free from disease as a result of physical examination alone, and therefore still more stress must be laid on the necessity for routine X-ray films before a negative diagnosis can be made. The logical consequence of this thesis is that chest radiology must be ranked with the other physical signs as an integral part of the examination of the chest.

The radical changes in the treatment of the acute respiratory infections which have resulted from the introduction of penicillin, and the multiplication of the drugs of the sulphonamide group, have necessitated the revision of those sections which deal with the treatment of acute infections.

JAMES MAXWELL

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# CONTENTS

## SECTION I

### THE HISTORY

| CHAP   | PAGE |
|--|------|
| 1. THE SIX MAJOR SYMPTOMS . . . . .          | 1    |
| 2. THE SIX MAJOR SYMPTOMS (contd.) . . . . . | 12   |
| 3. MINOR SYMPTOMS . . . . .                  | 21   |
| 4. THE AUXILIARY HISTORY . . . . .           | 26   |

## SECTION II

### PHYSICAL EXAMINATION OF THE CHEST

|                                     |    |
|-------------------------------------|----|
| 5. INSPECTION . . . . .             | 29 |
| 6. PALPATION . . . . .              | 38 |
| 7. PERCUSSION . . . . .             | 41 |
| 8. AUSCULTATION . . . . .           | 45 |
| 9. RADIOLOGY OF THE CHEST . . . . . | 52 |

## SECTION III

### SPECIAL INVESTIGATIONS

|  |    |
|--|----|
| 10. THE SPUTUM . . . . .                       | 71 |
| 11. BLOOD EXAMINATIONS—PLEURAL FLUID . . . . . | 85 |
| 12. SPECIAL DIAGNOSTIC METHODS . . . . .       | 89 |

## SECTION IV

### DISEASES OF THE RESPIRATORY TRACT

|  |     |
|--|-----|
| 13. THE UPPER RESPIRATORY TRACT . . . . .      | 92  |
| 14. THE LARYNX . . . . .                       | 101 |
| 15. THE TRACHEA AND BRONCHI . . . . .          | 108 |
| 16. ASTHMA . . . . .                           | 117 |
| 17. BRONCHIECTASIS . . . . .                   | 130 |
| 18. TUMOURS OF THE BRONCHI AND LUNGS . . . . . | 138 |



| CHAP.  | PAGE |
|--|------|
| 19. CREST INJURIES . . . . .                           | 149  |
| 20. THE PULMONARY CIRCULATION . . . . .                | 152  |
| 21. EMPHYSEMA . . . . .                                | 159  |
| 22. COLLAPSE OF THE LUNG . . . . .                     | 164  |
| 23. PNEUMONIA . . . . .                                | 172  |
| 24. BRONCHOPNEUMONIA . . . . .                         | 187  |
| 25. LUNG ABSCESS . . . . .                             | 197  |
| 26. PULMONARY TUBERCULOSIS . . . . .                   | 205  |
| 27. PULMONARY TUBERCULOSIS ( <i>contd.</i> ) . . . . . | 213  |
| 28. TREATMENT OF PULMONARY TUBERCULOSIS . . . . .      | 236  |
| 29. OTHER CHRONIC LUNG INFECTIONS . . . . .            | 257  |
| 30. FIBROSIS OF THE LUNG . . . . .                     | 259  |
| 31. THE PLEURA . . . . .                               | 265  |
| 32. "PLEURISY" . . . . .                               | 272  |
| 33. EMPYEMA . . . . .                                  | 283  |
| 34. TUMOURS OF THE PLEURA . . . . .                    | 289  |
| 35. THE MEDIASTINUM . . . . .                          | 290  |
| 36. THE DIAPHRAGM . . . . .                            | 299  |
| INDEX . . . . .  | 303  |

# LIST OF ILLUSTRATIONS

## (AT END OF VOLUME)

FIGS.

1. The author's pneumothorax apparatus
2. Chandler's modification of the Lillingston-Pearson pneumothorax apparatus
3. Normal chest
4. Scoliosis
5. Accessory lobe of the azygos vein. the vertebral borders of the scapulae are seen
6. Eventration of the diaphragm
7. Normal right bronchogram, anterior view
8. Normal right bronchogram, lateral view
9. Apparently normal chest, sputum contained tubercle bacilli
10. Tomogram of same case as fig. 9 showing cavity in left upper lobe
11. Large right-sided pleural effusion
12. Right-sided artificial pneumothorax
13. Massive collapse of the left lung; carcinoma of the left main bronchus
14. Dense fibrosis of right lung, chronic tuberculosis
15. Congenital cystic disease, right upper lobe
16. Congenital cystic disease, right upper lobe, bronchogram taken with patient supine
17. Congenital cystic bronchiectasis, left upper lobe, patient erect
18. Abscess in right lower lobe, lateral bronchogram. Lipiodol does not enter cavity; fluid level visible
19. Acquired bronchiectasis, left lung
20. Bronchiectasis in right lower lobe, lateral view
21. Atelectasis of left lower lobe; emphysematous appearance in left costophrenic sinus
22. Obstructive collapse of right lower lobe, bronchial carcinoma
23. Lung abscess showing fluid level (see fig. 24)
24. Lung abscess in right middle lobe (see fig. 23); lateral view
25. Abscess in right upper lobe, following tonsillectomy
26. Pneumonitis ("Primary atypical pneumonia")
27. Bronchial carcinoma, tumour in right hilum

it may be brought on or aggravated by change of posture. Certain special characters may also be of diagnostic value, pointing to the presence of a particular condition.

Cough may result from infection, from mechanical irritation, or from some reflex cause.

*Acute infections.*—The illness is of sudden onset and cough is often the presenting symptom. In these cases the cough is hard and dry at first, later productive. The most common are *laryngitis*, *tracheitis*, and *bronchitis*, all of which tend to be benign and are not accompanied by much constitutional disturbance. More serious are infections of the lung tissue, *pneumonia*, *bronchopneumonia*, and *lung abscess*, but in these the constitutional symptoms predominate and the patient is usually acutely ill. In senile patients, however, there may be little apparent general disturbance with quite extensive consolidation of the lungs, whereas, in very young children, even the mildest respiratory infection is apt to be associated with severe general symptoms, and in such cases it may be difficult to distinguish clinically between a simple bronchitis and bronchopneumonia.

In some cases, especially in children, a distinctive cough may be noted, consisting of a series of short, violent, expiratory efforts. This should always bring to mind the probability of *whooping-cough*, which can thus be recognized with some certainty several days before the characteristic "whoop" appears.

*Chronic infections.*—The respiratory passages, from the nose to the bronchioles, are very susceptible to infection, and repetition may lead to the development of a chronic inflammation. *Chronic bronchitis* and *chronic laryngitis* are the two main examples, but the upper respiratory tract commonly shares in the infection, a fact which is recognized in the term "broncho-sinusitis disease", coined in the United States to denote this condition. The mucous membrane of the whole tract is abnormal in these cases and a factor of mechanical irritation plays an important part in causing the cough, which is frequently unproductive.

Chronic cough may be a prominent symptom in *bronchiectasis*, although it is not such an essential part of the clinical picture as was at one time imagined, and it is not uncommon for patients with this disease to be free from all symptoms for long periods. On the other hand, when there is considerable

secretion stagnating in the dilated bronchi, it is sometimes noted that paroxysms of cough are brought on by change of posture. A history of cough, *aggravated by change of position*, is always suggestive of some form of cavitation with plentiful secretion, usually *bronchiectasis*, *lung abscess*, or *breaking-down growth*.

A very common chronic infection in which cough may be an early symptom is *pulmonary tuberculosis*. It is never safe to pronounce a definite opinion on any case of chronic chest disease until the sputum has been properly examined and the other appropriate investigations carried out, even in the absence of suspicious physical signs in the chest. In cases of pulmonary tuberculosis there are usually concomitant symptoms which suggest that the condition is more than a simple catarrh, yet this is by no means always the case and the only certain method of excluding the presence of tubercle is to search carefully for it. Other chronic lung infections are relatively infrequent.

*Mechanical irritation.*—Acute bronchial irritation, characterized by severe spasmodic cough, may result from the inhalation of irritant gases, in which cases the history usually makes the diagnosis clear, or from the lodgement of a foreign body either in the larynx or in a bronchus. The latter possibility should always be kept in mind when there is an abrupt onset of paroxysmal cough, especially in children, for a correct history is not always forthcoming, yet an early diagnosis may be crucial in determining recovery.

Chronic irritation, due to an abnormally sensitive mucous membrane, is responsible for the non-productive cough which is characteristic of *chronic bronchitis*, the commonest cause of persistent cough. Infection plays a part, but the cough is chiefly brought on by mechanical factors such as exposure to east winds, dusty atmosphere, and the inhalation of tobacco smoke; 'smoker's cough' is common, but it must be remembered that smokers may suffer from diseases of the respiratory tract which are not due to this habit. Chronic *sinusitis* may cause secretion to fall from the naso-pharynx on to the larynx, causing cough which wakes the patient during the night and is troublesome in the early morning. Over-use of the voice, as in clergymen, public speakers, and street hawkers, may be a contributory factor.

*Catarrh* is a condition which is very common and which may

may cause severe headache. Cerebral haemorrhage is an occasional sequel. Digestive symptoms, anorexia, vomiting, and consequent loss of weight, may all result from cough. The strain of constant coughing may even lead to serious physical exhaustion. It is important to recognize the secondary effects of the act of coughing and to associate these symptoms with their cause, for otherwise attention may be divided between different systems of the body when one is primarily at fault.

### Sputum

Inquiry should always be made about the *quantity and appearance* of the sputum and the *manner of its production*. The patient will generally be able to provide useful information on these points, although this must never be regarded as a sufficient substitute for the personal inspection of the specimen and the proper investigation which will be described in a later section (p. 71).

When there is irritation of the respiratory tract the sputum is, as a rule, scanty, clear, and viscid. With severe cough the presence of streaks of blood may not have any special significance. There is little sputum in cases of spasmodic asthma but, at the end of the attack, a small quantity of sticky secretion is usually voided.

The presence of yellow (*i.e.* purulent) sputum is proof of infection somewhere in the respiratory tract, but the colour is not a reliable indication of the type of organism present. In acute infections the secretion is scanty and tenacious at first; thus in the early stages of acute bronchitis and pneumonia a considerable effort of coughing will produce little result, although the infective nature of the illness will be disclosed by the constitutional symptoms. In the later, productive, stages of these infections the quantity of sputum increases and the cough is correspondingly looser. In chronic bronchitis the quantity varies greatly from time to time. There is often associated sinus infection, and not all of the "sputum" comes from the lower respiratory tract. A characteristic feature is that the cough and sputum are most marked in the early morning and, in mild cases, there is little sputum during the day. In pulmonary tuberculosis the amount varies greatly; at times there may be little or none, yet later there may be

several ounces and sometimes a large amount. Very large quantities of sputum may be produced in two conditions, *bronchiectasis* and *lung abscess*, although this symptom is not necessarily present in either. A large amount of pus coughed up suddenly is suggestive of the rupture of a lung abscess, *empyema*, or subphrenic abscess, into a bronchus. A rare condition is *bronchorrhoea serosa*, in which enormous quantities, up to several pints, of watery sputum may be evacuated on occasion. The exact nature of this condition is not clear. A greyish or black sputum signifies inhaled smoke, soot, or dust, and therefore indicates the presence of a mechanical irritant. Jet-black sputum ("black spit") is met with in anthracosis.

A very distinctive type of sputum is that in which fibrinous casts of the smaller bronchi are expectorated in a very rare condition, fibrinous bronchitis; occasionally, with pulmonary haemorrhage, somewhat similar bronchial casts composed of fibrin may be coughed up.

The physical attributes of the sputum which may be apparent to the patient are those of taste and smell. It is not uncommon for the viscid muco-pus of acute bronchitis to be observed to possess a distinctly sweet taste. A foul taste and smell are often present in cases of *bronchiectasis*, *lung abscess*, and *malignant growth*. It is curious to note that the patient is often less aware than are his companions of the offensive nature of the sputum.

Information may be gained by inquiry about the effect of *change of position* on the quantity of sputum. In any condition in which large cavities are present, notably in *bronchiectasis* and *lung abscess*, the patient may state that sudden movement, either from side to side, or from supine to erect, or vice versa, is followed by vigorous coughing and copious expectoration. A history to this effect is very suggestive of one of these conditions, and indicates the probable value of postural methods in securing drainage.

Finally, specific inquiry must always be made about the presence of blood in the phlegm. Many patients are unduly reticent on this point, and haemorrhage may be wrongly assumed not to have occurred because it is not voluntarily mentioned, yet the fact that there has been blood in the sputum may be of the utmost diagnostic importance and a definite statement should always be obtained on this point.

## Haemoptysis

This is perhaps the most important symptom of all, for blood-spitting nearly always indicates the presence of serious disease in the respiratory tract. But first it is necessary to be sure that the blood really has been coughed up and that it does not come from some other source. The common history is that, with a slight cough, or even without any cough at all, there is a warm salt taste at the back of the throat, and this is followed by the expulsion of blood. The blood which comes from the respiratory tract is bright and frothy as a rule, and perhaps the most important diagnostic point is that *the sputum contains streaks of blood for some days after the initial haemorrhage*. The common sources of extraneous bleeding are the nose, the mouth, and the stomach. Nose-bleeding is usually obvious, for blood nearly always escapes by the anterior nares as well, but, in doubtful cases, proper examination of the naso-pharynx must be undertaken. Occasionally, especially in children, an unhealthy spongy condition of the gums may give rise to sufficient bleeding to cause staining of the pillow after the patient has been asleep, and it is obviously important to recognize the source of the blood in such cases. Bleeding from dilated veins at the back of the throat is sometimes held to account for haemoptysis, and doubtless such a diagnosis is occasionally correct, but the presence of an extraneous source for apparent haemoptysis must never be assumed except on the clearest evidence, and in all cases the state of the lower respiratory tract must be thoroughly investigated. Malingerers have found the pharyngeal veins a useful source of blood, and in such cases a diagnosis can only be made after a period of careful observation. Haematemesis is sometimes confused with haemoptysis, and the patient may be uncertain whether the blood is coughed or vomited; blood from the stomach is often clotted and mixed with food residue, and an acid reaction to litmus is strong evidence of a gastric origin.

Haemoptysis may be either mild or severe. In the former case there may be simply a few streaks of blood in the sputum, or perhaps a drachm of blood at times; this is the type of haemorrhage which occurs in *early pulmonary tuberculosis* and results from leakage from dilated capillaries. It may also occur in acute or chronic bronchitis, in asthma, and in

whooping-cough, but its presence should always be regarded with suspicion.

More profuse haemoptysis occurs most commonly in the later stages of *tuberculosis*, *bronchiectasis*, *bronchial carcinoma*, and in heart disease, especially with *mitral stenosis*. The loss of blood may vary from a few ounces to a pint or more. In the later stages of *pulmonary tuberculosis* severe haemorrhage is due to the rupture of a small aneurysm of a branch of the pulmonary artery lying in a cavity, and it is sometimes rapidly fatal. The haemorrhagic form of *bronchiectasis* is a commoner cause of haemoptysis than is usually realized, and any case in which tubercle can be confidently excluded should be examined with lipiodol in order to determine the presence of this condition.

Haemoptysis is a common symptom of *bronchial carcinoma*, and this possibility should be borne in mind, especially in patients of middle age.

In heart disease haemoptysis occurs in two main types of condition. In *mitral stenosis infarction* may result from thrombosis of a branch of the pulmonary artery or of a tributary of the pulmonary vein; the bleeding is not copious and is accompanied by pain in the chest. The most dramatic haemoptysis of all occurs when an aneurysm of the aorta ruptures into a main bronchus with, as a rule, immediately fatal results.

Classification of the causes of haemoptysis is best carried out on an anatomical basis.

### CAUSES OF HAEMOPTYSIS

#### *Local causes in the respiratory tract.*

##### (a) *Larynx.*

(i) Injury.

(ii) Laryngitis.

Acute (rare).

Tuberculosis.

(iii) Malignant growth.

##### (b) *Trachea.*

Lesions very rare but growth does occur.

##### (c) *Bronchi.*

(i) Foreign body.

(ii) Acute inflammation, whooping-cough, acute bronchitis (rare).

(iii) Chronic inflammation, *bronchiectasis*, chronic bronchitis.



- (iv) *Tumour. Bronchial carcinoma*; innocent tumour.
- (v) Erosion of a bronchus by aneurysm.
- (d) *Lungs.*
  - (i) Injury, penetrating wounds.
  - (ii) Vascular causes, *infarction* (usually with mitral stenosis), pulmonary embolism, congestion of the lungs.
  - (iii) Acute infections, *pneumonia, lung abscess.*
  - (iv) Chronic infections, *pulmonary tuberculosis*, others rare.
  - (v) Infection by parasites, lung fluke, hydatid, amoebic abscess.

*General causes (all rare).*

- (a) Disorders of the blood-forming tissues, purpura, acute leukaemia.
- (b) Haemorrhagic forms of acute specific fevers.
- (c) Haemophilia and scurvy.

In certain conditions the blood is altered in character before it is coughed up, and the appearance of the sputum may be of great help in forming a diagnosis. In lobar pneumonia, for instance, it is usually tinged with altered blood of a bright brownish-yellow colour, forming the classical "rusty sputum" of this condition. In lung abscess and in bronchiectasis the blood may be intimately mixed with pus to form a thick, pinkish liquid, although in each of these conditions there may be frank haemorrhage on occasion.

It must be admitted that, so far as our investigations will take us, we are left with a considerable residue of cases in which there has been a proven haemoptysis for which no cause can be found. In some of these cases there is a rapid recovery of health and no subsequent breakdown; although the safe course is obviously to treat the condition as one of early tuberculosis in the absence of evidence to the contrary, there must always remain some doubt about the nature of these cases.

It is customary to doubt the existence of haemoptysis occurring as a manifestation of vicarious menstruation, although this condition has often been described. Such cases undoubtedly occur, although they are rare. It is probable that vicarious bleeding does not occur from a normal respiratory tract.

#### INVESTIGATION OF HAEMOPTYSIS

The routine investigations to be adopted in the average case should run on the following lines:—

(i) The *history* is likely to yield evidence of the presence of respiratory or of cardio-vascular disease. Previous cough, sputum, night sweats, and loss of weight are very suggestive of *tuberculosis*; repeated severe haemoptyses without obvious deterioration are in favour of *bronchiectasis*. A haemoptysis occurring for the first time in a patient of middle age is suspicious of *malignant growth*. In *pulmonary infarction* the history may be that of old rheumatic heart disease, of phlebitis, or of recent operation, and the attack usually coincides with acute pain in the chest.

(ii) Due importance must always be attached to the *family history*, for haemoptysis occurring in a patient, one or more of whose relations are known to be tubercular, must be suspected of being due to this cause until proof to the contrary has been obtained.

(iii) *Physical examination* must be directed separately to the state of the lungs and of the heart. Yet, no matter what physical signs are present, the appropriate special investigations must always be carried out in order to determine the cause with certainty.

(iv) An evening temperature record should be kept in all cases in which there is the slightest reason to suspect the presence of tuberculosis.

(v) *The sputum must be examined carefully and repeatedly* in all cases for tubercle bacilli, no matter how little the clinical condition appears to point to tuberculosis. Search for parasites should also be undertaken whenever the circumstances appear to warrant this step.

(vi) *An X-ray of the chest should be taken in all cases*, even when there is clinical evidence of heart disease.

(vii) If the sputum is repeatedly negative for tubercle, and if the X-ray is inconclusive, a *lipiodol examination* should next be carried out in order to demonstrate the presence of bronchial dilatation, or of obstruction due to growth.

(viii) In some cases it will be desirable to examine the larynx, both for evidence of tuberculous ulceration and to determine the mobility of the vocal cords.

(ix) Finally, a *bronchoscopy* is often necessary in order to exclude foreign body and malignant growth.

The significance of haemoptysis has been considered in some detail because it is one of the most important symptoms of chest disease, and because it is frequently underestimated, with disastrous results to the patient who happens to be suffering from early tuberculosis or growth. If, however, the symptom is regarded seriously and investigated in the manner suggested, mistakes in diagnosis will be comparatively rare.

## CHAPTER 2

### THE SIX MAJOR SYMPTOMS (*continued*)

#### Thoracic Pain

LUNG tissue is insensitive, and pain in the chest results from involvement of surrounding structures. *The pain of respiratory disease is nearly always unilateral.*

#### SITES OF PAIN IN THE CHEST

- (a) *The pleura.*
  - (i) *Pleurisy.*
    - Acute.
      - "Primary" (usually tuberculous).
      - Secondary to lung disease.
      - Infarct.
    - Chronic.
      - Inflammatory.
      - Neoplastic.
  - (ii) *Sudden alteration in intra-pleural pressure.*
    - Spontaneous pneumothorax.
    - Massive collapse of the lung.
- (b) *The chest wall.*
  - (i) *The ribs.*
    - Injury.
    - Inflammation, acute or chronic.
    - Growth (primary or secondary).
    - Erosion.
  - (ii) *The muscles and fibrous tissues.*
  - (iii) *The breasts.*
- (c) *The sensory nervous system.*
  - (i) *The spinal cord and posterior roots.*
    - Herpes zoster.
    - Tumours, meningeal and vertebral.
    - Pott's disease.
  - (ii) *The intercostal nerves.*
    - Neuritis.
    - Neuralgia.
  - (iii) *Psychogenic ("pain of reminiscence").*
- (d) *The heart and aorta.*
  - (i) Pericarditis.
  - (ii) Acute ventricular dilatation.
  - (iii) Coronary disease.
  - (iv) Aortic disease.

It is essential to locate the origin of chest pain and all of these possibilities have to be considered. The pain which originates in the pleura is rarely sharply defined and it is usually *worse on inspiration*. Pain in the chest wall, on the other hand, is often localized, and *tenderness* is noticed on palpation. A very common source of diagnostic error is the pain which is due to involvement of the sensory nerve supply, for one is apt to forget the spinal cord and its connections and to concentrate on the physical signs in the chest. Pain which radiates along the course of one or two intercostal nerves usually has its origin in some part of the local sensory apparatus. Cardiac pain is commonly precordial, whereas respiratory pain nearly always affects one side of the chest, but an abnormal distribution of either is an occasional cause of confusion.

(a) *The pleura.*—*Fibrinous pleurisy* causes severe pain, *worse on inspiration*. It commonly occurs as a complication of pneumonia, but it may be "primary", in which case it is often due to tuberculosis even although there may be no evidence of disease in the underlying lung. Infarct usually occurs just beneath the pleura, causing an aseptic pleurisy which can be very painful. The pain of dry pleurisy is aggravated by lying on the affected side and relieved by lying on the sound side.

When the diaphragmatic pleura is inflamed the pain may be referred to the abdomen, and an acute chest condition may closely simulate perforated gastric ulcer or acute appendicitis. In all cases of apparent abdominal emergency it is necessary to be sure that the base of the lung is clear before deciding on operation. Acute lesions of either surface of the diaphragm may also cause referred pain in the shoulder as a result of irritation of the phrenic nerve.

When the inflammation extends a stage further, exudation, either of clear fluid or of pus, results and the acute pain is replaced by a dull ache. It is little affected by inspiration and is lessened by lying on the affected side. The severity of this pain is in proportion to the rate at which the fluid accumulates.

Chronic pleurisy implies the presence of pleural adhesions and there is surprisingly little pain as a rule; it is rarely wise to make this diagnosis, especially in the hearing of the patient, unless there are ample clinical grounds on which to base it. Invasion of the pleura by *malignant growth*, either primary or secondary, is sometimes marked by severe pain.

Normally the pleural surfaces are in apposition and there is a slight negative tension in the pleural space. Any sudden alteration in the pressure, in either direction, may cause acute pain. A *spontaneous pneumothorax* may cause only a dull ache unless there is a rapidly rising intrapleural pressure, in which case the pain may be quite severe; if there are areas of adhesion, the separation of the layers of pleura may cause dragging pain in the affected area. Conversely, the sudden absorption of air from the lung in cases of *massive collapse* causes an abrupt increase in the tension, with displacement of the mediastinum and diaphragm, and acute pain. A similar result may ensue when a pleural effusion is evacuated too rapidly.

(b) *The chest wall.*—Injury and inflammation of ribs cause local pain and tenderness, but they should present little diagnostic difficulty. Erosion of the ribs or sternum, whether by aneurysm or by malignant growth, results in severe localized pain often described as “boring” in character; X-ray examination should make the diagnosis clear. Acute inflammation of the fibrous tissues and muscles of the chest wall, commonly termed “fibrositis”, is a quite common cause of pain and tenderness. Finally, it should not be forgotten that disease of the breast is a possible cause of pain in the front of the chest.

(c) *The sensory nerve supply.*—Intercostal nerve pain radiates along the course of one or more nerves and may be severe; the distribution and the fact that it is not affected by respiration are sufficient to identify this type. The cause may be intercostal neuritis or neuralgia, but care must be exercised in making this diagnosis for a similar nerve pain may result from other causes; the pain of *herpes zoster* may precede the appearance of the rash by several days. Growth may be present in the vertebrae or in the spinal meninges, and the possibility of *tuberculous caries* must also be kept in mind. Nodules of secondary growth in the spine are usually apparent in an X-ray; a careful search for a primary growth anywhere in the body, but especially in the breast, bronchi, stomach, prostate, kidneys and thyroid gland, is necessary in every suspicious case.

A label of “pleurodynia” is sometimes applied when no organic explanation of the cause of the pain is forthcoming. The term simply indicates that there is a pain in the chest and it usually means that the cause of the pain has been overlooked.

Patients are sometimes met with who complain of severe chest pain which has been more or less continuous since a genuine attack of pleurisy months or years before. In these cases there is no organic explanation to be found and it can only be that the persistence of this symptom has a psychological foundation. This pain may well be termed a "pain of reminiscence", for it appears to be the result of a persistent mental picture of the original illness. The patient is usually temperamental, often obsessed with the notion that the pain is due to the presence of pleural adhesions, and the condition does not respond well to treatment.

(d) *The heart and aorta.*—Pain which results from heart disease is primarily precordial and there is seldom difficulty in distinguishing cardiac from respiratory pain. Acute fibrinous pericarditis causes a localized sharp pain and acute dilatation of the ventricles causes a more diffuse severe ache, but both are precordial and they do not vary with respiration. Valvular lesions do not of themselves cause pain.

Disease of the coronary arteries is traditionally associated with *angina*, a severe precordial pain which often radiates along the inner border of the left arm. The left upper chest is frequently affected. A *coronary thrombosis* is a major catastrophe, with severe pain in the precordia and in the epigastrium.

Atheroma of the aorta is commonly associated with disease of the coronary vessels in elderly subjects. The pain is anginal in type. *Aneurysm* may give rise to a similar pain but it may also cause a severe localized aching or boring pain when bone is being eroded.

(e) *Referred pain*—Inflammation or growth in connection with the diaphragm is sometimes difficult to identify because the pain is likely to be referred to a distance from the site of the trouble. Lesions of the posterior part of the diaphragm are associated with pain in the lumbar region, lesions of the part in contact with the parietal pleura cause pain in the side of the chest, while central lesions are responsible for pain in the neck and shoulder.

Disease of the gall-bladder is well known to cause pain referred to the right shoulder; it is said, although this is less well authenticated, that lesions of the stomach may be associated with pain in the left shoulder. In all cases of obscure chest pain where there are no signs of disease above the

diaphragm it is necessary to consider the possibility of some upper abdominal condition being the cause of the symptom.

### Dyspnoea

The term literally means "difficult breathing" and can be defined as consciousness of the need for increased respiratory effort. *Hyperpnoea* simply means increased depth of breathing, while *tachypnoea* indicates an increase in the respiratory rate.

The *vital capacity* is an index of the functional efficiency of the respiratory tract. The amount of air taken in on average inspiration is between 350 and 500 c.cs., this being known as *tidal air*. On forced inspiration a further 1500 c.cs. can be taken in, *complemental air*. The largest volume of air which can be expelled by the most vigorous exhalation, made at the end of an ordinary expiration, amounts to between 1000 and 1500 c.cs., and this is known as *supplemental air*. The total volume of air which can be taken into and expelled from the lungs by the most forcible inspiration and expiration, i.e. the sum of the tidal, complemental, and supplemental air, constitutes the *vital capacity*, which is therefore normally from 3 to 3½ litres. It may be necessary to determine this figure in order to decide whether a patient is fit to withstand one of the major chest operations. Even after the most forcible expiration a considerable quantity of air, about 1000 c.cs., still remains in the lungs and this is termed *residual air*.

### CAUSES OF DYSPNOEA

*Respiratory obstruction.*

*Reduction of the aerating surface of the lung.*

*Disturbances of the respiratory centre.*

*Interference with the pulmonary circulation.*

*Respiratory obstruction.*—Acute obstruction in the larynx may result from *foreign body, strangulation, diphtheritic membrane, or oedema*. In these cases the patient is acutely ill, and the history and the examination of the throat should suffice to establish the diagnosis.

Obstruction of the lumen of the trachea is uncommon, but growths occur occasionally. External pressure from malignant tumour of the thyroid gland or other swellings in the neck is rare; the usual site of tracheal compression is the confined

space behind the manubrium sterni, in which a relatively small tumour or aneurysm may cause severe pressure symptoms.

*Reduction of the aerating surface of the lung.*—In acute conditions the degree of dyspnoea is proportional to the rate of deflation of the lung. A rapidly accumulating *pleural effusion*, a sudden *spontaneous pneumothorax*, or an acute *massive collapse of the lung* may cause urgent dyspnoea, whereas the same conditions occurring gradually may be almost symptomless. A sudden shift of the mediastinum appears to be the factor which determines the severity of the dyspnoea.

It is well known that a life of normal activity can be carried on although one lung is not functioning at all, always provided that the change has occurred sufficiently slowly. Patients with complete collapse of a lung due to growth, with extensive fibrosis of one lung, or even following a total pneumonectomy, may not be at all dyspnoeic on moderate exertion. But degrees of lung destruction which are more extensive, affecting two-thirds or more of the ventilating capacity, render the patient increasingly short of breath. The most common cause of this kind is *emphysema*, but extensive *bilateral fibrosis of the lungs* such as occurs in *silicosis*, in *asbestosis*, and in advanced *fibroid tuberculosis*, may cause severe and progressive dyspnoea.

*The respiratory centre.*—Respiration is controlled by the respiratory centre, and this, in turn, is influenced by a variety of factors, of which the most important are nervous impulses from the vagus and the higher centres and chemical changes in the blood. Increase in the H-ion concentration, usually from excess of carbon dioxide but sometimes from accumulation of lactic or other organic acids, results in increased depth of breathing. The "tendency to acidæmia" thus brought about is responsible for the dyspnoea seen in *uraemia* and in some cases of heart-failure. In the *ketosis of diabetes* the "enol" bodies in the blood exert a specific effect on the centre, giving rise to "air-hunger". Oxygen lack causes rapid, shallow breathing, and also periodicity of the respiratory rhythm. Its action appears not to be directly on the centre, but on the nerve endings in the carotid sinus and the aortic arch, setting up excitatory impulses which reflexly stimulate breathing. A degree of dyspnoea is present at *high altitudes* for this reason; when the ventilating surface is diminished a similar effect will be noted at proportionately lower levels.



It would appear that the dyspnoea which is such a characteristic feature of *lobar pneumonia* and of *bronchopneumonia* is really central in origin. The degree of dyspnoea bears no relation to the extent of lung involvement, and it can only be due to the toxæmia which is the striking feature of the disease. In *spasmodic asthma* it is well known that the essential feature is a disturbance of respiratory control through the centre. Nervousness often causes the respiratory rate to be increased, and true *hysterical hyperpnoea* and *tachypnoea* may occur.

*Interference with the pulmonary circulation.*—Sudden interference with the blood supply of a considerable part of the lung, such as occurs in *pulmonary embolism*, may cause intense dyspnoea; in such cases the diagnosis may be easy if a source of embolism is known to be present, but it may be very difficult if the picture is that of urgent dyspnoea without apparent cause. The pulmonary vessels themselves may be narrowed, as in *Ayerza's disease*, or there may be simple mechanical congestion, as occurs in right heart-failure. In these cases shortness of breath is a prominent feature.

The factors concerned in the dyspnoea of heart-failure are complex and vary according to the state of the heart and circulation and the extent of coincident lung damage. The chief factors are stated by Samson Wright to be (i) raised metabolic rate at rest; (ii) diminished vital capacity; (iii) anoxia and consequently a malnourished respiratory centre; (iv) carbon dioxide accumulation at times and acidaemia. Secondary renal failure may complicate the issue, causing the retention of non-volatile acids. A careful overhaul of the circulation as well as the lungs is therefore necessary whenever shortness of breath is a presenting symptom, for the two systems are so closely connected that, although the primary trouble may be in one, there may well be extensive changes in the other.

*Failure of the muscles of respiration.*—Acute dyspnoea may result from conditions which affect the muscles of respiration. Sudden paralysis of the diaphragm may cause great distress, and *abdominal distension* may interfere markedly with inspiration. The intercostal muscles are rarely paralysed, but in certain acute lesions of the spinal cord, for instance *acute poliomyelitis*, there may even be fatal dyspnoea.

*Minor factors.*—Of the minor factors concerned in some cases, increased metabolism, changes in the state of the blood,

either anaemia or polycythaemia, and chronic anoxaemia from the use of drugs should be kept in mind. These are not by themselves sufficient to cause marked dyspnoea, but they may accentuate the effect of the other factors which have already been considered. Obesity is also a common contributory factor, especially as it often occurs in those who already suffer from emphysema or from chronic heart disease.

Two other terms need to be defined. In severe degrees of dyspnoea the patient may only be able to breathe in an upright position, a state to which the name *orthopnoea* is given; in this position the inspiratory action of the diaphragm is materially assisted by gravity. The other abnormality is that known as *Cheyne-Stokes breathing*. This occurs in a variety of pathological conditions and is nearly always a serious symptom. The respiratory rhythm exhibits a *periodicity* characterized by a period of apnoea, after which the breathing recommences, being shallow at first and gradually increasing in depth until there is hyperpnoea; the excursions then become smaller and in a short time cease altogether, being succeeded by a further period of apnoea. This phenomenon is due to oxygen lack or to the action of poisons, usually acids, which accumulate in the blood stream. It most commonly results either from interference with the cerebral circulation, such as may occur in pontine vascular damage, tumour or abscess, or from heart-failure or uraemia. Cheyne-Stokes breathing is often first noticed when the patient is asleep.

### Weight

Change in weight ranks as a major symptom because it is an early, and sometimes the earliest, symptom of *tuberculosis*; also, the weight chart is a very reliable guide in assessing the progress of a patient under treatment.

The fact of a change in weight is emphasized, for a steady weight, either over or under the average, is not particularly suggestive of active disease. An individual may be thin and much underweight and yet may be in good health, nor is there any reason to consider that those who are naturally thin are therefore predisposed to develop tubercle. Those who are too fat are, perhaps, rather prone to develop chronic bronchitis and emphysema. Loss of weight is a cardinal symptom of active tuberculosis, as the term "*phthisis*" suggests, and the

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## CHAPTER 3

### MINOR SYMPTOMS

IN addition to the six major symptoms inquiry must be made about subsidiary symptoms. These may be referable directly to the respiratory tract, they may be general, or they may concern some other system, the function of which may have been affected as a secondary event.

#### Respiratory Symptoms

##### *Wheeze*

This is more pronounced on *expiration* and, if it can be definitely established that there is a respiratory wheeze, then it is almost certain that the symptom is due to bronchial spasm and is therefore likely to be *asthmatic*. In many cases of "bronchitis" in children wheeze is present and, in such, true spasmodic asthma frequently supervenes.

##### *Stridor*

This is sometimes confused with the foregoing. The noise produced is more strident and is chiefly *inspiratory*. Stridor has its origin in the larynx or trachea, and it therefore indicates obstruction somewhere between the vocal cords and the main bronchi. The common causes are *tumour*, *aneurysm*, or a *mass of glands*.

In infants and small children there are certain well-defined conditions which cause inspiratory stridor. As the personal history is unobtainable in these cases, the diagnosis must be made from a consideration of the clinical state (p. 103).

##### *Voice Change*

Loss of voice occurs with any laryngeal disease, or it may result from paralysis of a recurrent laryngeal nerve. Recurrent loss of voice may result from repeated attacks of laryngitis, but it sometimes precedes the development of frank pulmonary tuberculosis. Chronic huskiness is a feature of *chronic laryngitis*, although it may also occur in *tuberculosis*, *syphilis*, or *growth* of the larynx. *Aphonia* may also be hysterical in origin.

degree of weight loss is roughly proportional to the severity of the infection.

There may, however, be considerable loss of weight in many other respiratory conditions, notably in malignant disease, and also in severe bronchiectasis and spasmodic asthma. Of course, loss of weight occurring as a sudden event in the course of some chronic respiratory disease should always suggest the possibility of a secondary tuberculous infection.

Loss of weight is the one major symptom which is not chiefly concerned with conditions affecting the respiratory tract, for it commonly results from disease in most systems of the body. Especially in cases of early hyperthyroidism, the clinical resemblance to tuberculosis may be close and the differential diagnosis may have to be carefully considered. When loss of weight without other obvious clinical features is the presenting symptom the urine must be tested for sugar in order to exclude the possibility of diabetes mellitus.

Malignant disease in any part of the body is a common cause of loss of weight, and especially in cases of cancer of the oesophagus and stomach.

Organic digestive disturbances are obvious causes of weight loss. Gastric and duodenal ulcer, intestinal tuberculosis, and any type of colitis are common examples of non-malignant organic disease which may cause wasting, and it is frequently necessary to investigate the state of the intestinal tract by means of a barium X-ray, even in patients who are known to have disease in the chest. The patient may become very thin as a result of functional disorders of the digestion, such as achalasia of the cardia, atonic dilatation of the stomach, and anorexia nervosa. Finally, it must be remembered that voluntary attempts at "slimming" may result in excessive weight loss and the consequent loss of strength may easily render the subject liable to contract tuberculosis.

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*Upper Respiratory Catarrh*

A history of repeated colds frequently indicates infection of the nasal sinuses, which is a most important factor in causing chronic infection of the lower respiratory tract. In these cases the nose must be carefully overhauled. *Hay fever* and *paroxysmal rhinorrhoea* are commonly present in asthmatic patients, or they may occur separately; the attacks of paroxysmal sneezing are characteristic. Hay fever, of course, is strictly seasonal.

*General Symptoms*

General symptoms usually result from toxæmia and therefore occur in severe acute infections, such as pneumonia, or in chronic infections, notably tuberculosis. The severity of these symptoms is a rough clinical guide to the balance between the virulence of the infection and the resistance of the patient.

*Malaise*

A vague symptom which may indicate gross disease or a comparatively trivial ailment. It is common in all infections as well as in wasting disease. When it occurs without any localizing symptoms it is always desirable to examine the chest carefully from the point of view of latent tuberculosis and cancer.

*Undue Fatigue*

Perhaps the vaguest of all symptoms is fatigue. Especially at the present time it is very common for patients to complain of feeling unduly tired, and this may mean nothing in terms of organic disease. Yet fatigue is an early symptom of *tuberculosis* and *growth*, so that full investigation must always be undertaken whenever this symptom is prominent.

*Night Sweats*

This is a common complaint in *tuberculosis* and it serves as an indication of the severity of the disease. Less frequently it is noted in cases of *lymphadenoma* and in malignant states. The presence of night sweats does not necessarily indicate that the disease is situated in the respiratory system, although this is usually the case. In children marked sweating is often associated with active ricketts.

## Symptoms in Other Systems

*Dysphagia*

The chief causes of dysphagia, although intrathoracic, do not directly concern the respiratory tract, yet the respiratory causes are by no means unimportant.

The symptom may result from causes in the throat, such as acute tonsillitis, quinsy, mumps, and retropharyngeal abscess. *Tuberculosis of the larynx* causes much pain on swallowing, whereas *syphilis* leads eventually to a stricture at the upper end of the gullet. So far as the oesophagus itself is concerned, impacted *foreign body* and *carcinoma* are the common causes, and disorder of the neuromuscular control of the cardiac sphincter leads to the development of the condition known as *achalasia of the cardia* or, less correctly, as cardio-spasm.

Pressure on the oesophagus may occur in the neck or in the chest. Malignant disease of the thyroid gland and *exophthalmic goitre* are occasional causes of dysphagia, but *retrosternal goitre*, *intrathoracic tumour*, and *thoracic aneurysm* are commoner. Rarely, enlargement of the heart, or a pericardial effusion, may press sufficiently on the oesophagus to produce mild dysphagia.

*Dyspepsia*

Many respiratory disorders may cause digestive disturbance, either reflexly or as a result of toxic absorption. An initial complaint in *pulmonary tuberculosis* may be dyspepsia of the acid type, with pain after food. The correct diagnosis in such cases is easily missed at the critical stage of the disease. In the later stages *anajonic dyspepsia* may occur, shown by anorexia, nausea, vomiting, and loss of weight. These symptoms result from toxæmia and indicate advanced disease; they may also occur in bronchiectasis and in bronchial carcinoma.

When there is severe spasmodic cough, as in *whooping-cough*, mechanical vomiting after a bout of coughing is common.

In acute toxic conditions, such as *pneumonia*, the degree of digestive disturbance, as evidenced by anorexia, vomiting, and diarrhoea, is a measure of the severity of the toxæmia. In the most severe cases acute dilatation of the stomach may occur.



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There is a tendency for all mucous membranes to be affected in *catarrh*. It is quite common for a patient with post-nasal *catarrh* to complain also of symptoms suggestive of "gastritis" or of "colitis".

### *Intestinal Symptoms*

Diarrhoea is not uncommon in tuberculosis when the bowel has become ulcerated, and this symptom is therefore of grave significance in *pulmonary tuberculosis*. Toxic diarrhoea is a serious symptom in pneumonia. In chronic suppurative conditions diarrhoea is suggestive of *amyloid disease*, which may occur as a complication of bronchiectasis, lung abscess, chronic empyema, and, rarely, tuberculosis.

*Fistula-in-ano*, when it occurs without obvious cause, is suggestive of tuberculosis and is sometimes present considerably in advance of the actual respiratory symptoms. In such cases a careful overhaul of the chest is essential.

### *Amenorrhoea*

Sudden cessation of the periods is a not uncommon symptom in *early pulmonary tuberculosis*, and the occurrence of amenorrhoea with respiratory symptoms suggests the possibility of tubercle. It may also occur in other wasting conditions.

### *Skin Conditions*

These are rarely likely to be an initial complaint. In *allergic asthma* there is often an associated eczema or urticaria, in the patient or in the family. In *pulmonary tuberculosis* there may be pityriasis versicolor, a brownish red eruption seen as a rule on the shoulders and due to *microsporon furfur*. *Erythema nodosum* may precede phthisis; other tuberculides are rare. Cutaneous granulomata are sometimes seen in cases of fungus infection of the lung and in Boeck's sarcoidosis. Sometimes nodules of secondary growth are found in the skin of the trunk in cases of *bronchial carcinoma*.

It is quite common, in patients of middle age, to find small, slightly raised, deep-red spots on the trunk. These are known as Campbell de Morgan's spots; they do not fade on pressure, being due to capillary telangiectases, and they are of no significance, except that they must not be mistaken for a purpuric rash.

### *Joint Conditions*

Acute suppurative arthritis is an occasional complication of pneumonia. A diffuse form of polyarthritis, apparently toxic in origin, may occur in *pulmonary tuberculosis*. This condition has been described in the French literature and it is important to keep it in mind, for sometimes there are no chest symptoms and the focal infection is assumed to be streptococcal. Very marked swelling of the medium-sized joints, occurring with clubbing of the fingers, and known as *hypertrophic pulmonary osteoarthropathy*, is sometimes seen in cases of *chronic suppuration*, *pulmonary syphilis*, and *bronchial carcinoma*.

### *The Nervous System*

In acute infections of the respiratory tract there may be toxic delirium which may persist for some time after the cause has ceased to operate. A toxic psychosis is an occasional complication of pneumonia but it is rarely persistent. Meningismus is not uncommon, especially in the apical pneumonia of children, but true meningitis is fortunately a rare complication. There appears to be a curious connection between lesions in the lung and in the brain, for *lung abscess* and *bronchiectasis* are common causes of cerebral abscess, and *bronchial carcinoma* frequently gives rise to cerebral metastases. In fact, a primary cerebral tumour should not be diagnosed until it is certain that the lungs are clear.

The spinal cord may be affected by pressure on the vertebrae by *aneurysm* or *growth*, or by permeation of the foramina by *intrathoracic tumour*. These conditions must be kept in mind when investigating the cause of a spastic paraplegia.

The peripheral nervous system is rarely affected, but an acute polyneuritis sometimes complicates pneumonia.

In conclusion, it must be remembered that advanced chest disease may be present with few or no symptoms. A bronchial carcinoma may give rise to metastases anywhere in the body, and the secondary growths may cause symptoms while the primary is yet latent; for instance, a metastasis in the brain may lead to an erroneous diagnosis of a primary cerebral tumour. Even in the absence of respiratory symptoms it is always as well to take all precautions to exclude disease in the respiratory tract.

## CHAPTER 4

### THE AUXILIARY HISTORY

#### The Past History

THE past life of the individual is often reflected in the present clinical state, so that a knowledge of the background and of the habits is often essential, not only in diagnosis but also in prognosis. Consideration of the previous illnesses is also important, for the present condition may be a sequel of past disease.

It is necessary to know the life the patient is accustomed to lead, the amount of travelling that he has to do, the atmosphere both at work and in the home. Over-work, mental worry, fatigue, poor nutrition, and bad ventilation are potent factors in diminishing resistance to infection and thus are likely precursors of tuberculosis. Injudicious dieting in order to reduce weight reduces resistance as well, as does excessive sun-bathing in those who are not suited to it. Those who eat too much, and who are out of condition in consequence, are prone to develop chronic bronchitis and emphysema, and their gross habit spells poor resistance to infection, so that the chance of recovery from pneumonia is less than average. Alcoholics are also bad risks so far as chest infections are concerned. Tobacco smoking may cause irritation of the upper respiratory tract and thus may be responsible for the persistence of cough in pharyngitis and in laryngitis. *It has been shown that heavy smokers are more liable to develop post-operative collapse of the lung.*

Recent injury is a possible cause of such acute conditions as haemothorax and pneumothorax, or it may explain a haemoptysis. An injury can also aggravate or reactivate some pre-existing condition. But injury cannot cause tuberculosis, nor is there any evidence that it can be responsible for the development of malignant disease.

The occupation must next be considered. - There are some respiratory diseases which are due to the inhalation of dust, such as *silicosis* and *asbestosis*, but there is no evidence that the inhalation of irritant dust plays any part in causing bronchial

cancer in this country, although the tendency of the cobalt miners of Schneeberg to develop this disease is well known. It is not sufficient to inquire about the present occupation, for this may have been recently changed, and it is better to obtain a brief summary of the life of the individual. For instance, a patient who describes himself as a "night watchman" has almost certainly pursued an entirely different occupation in the past. Bronchitis may be aggravated by the atmosphere in which a patient works, when it is either dusty or steamy. A patient may become sensitive to some foreign protein which he handles in the course of his work and he may develop asthma.

The mechanical effect of certain occupations on the lungs is well recognized. Glass-blowers are said to develop emphysema; it is less certain that players of wind instruments suffer in this way.

The part played by previous disease must next be considered. Of the acute specific fevers, *measles* and *whooping-cough* predispose towards *pneumonia* as a complication and to *bronchiectasis* as a sequel. A history of repeated attacks of "pneumonia" is commonly found in cases of bronchiectasis and in asthmatics, in whom the acute condition is not always a true pneumonia. Operation on the upper respiratory tract, such as the removal of tonsils, teeth, or nasal polypi, is sometimes followed by *lung abscess*. Previous pleurisy, either dry or with effusion, must be regarded as a strong point in favour of a diagnosis of *tuberculosis* in a young adult who presents respiratory symptoms, and a history of *erythema nodosum* has a like significance. It is common to obtain a story of repeated attacks of "influenza"; this diagnosis is so simple that it is commonly wrong, except in epidemics. In cases of sporadic influenza it is necessary to watch the state of the lungs very carefully for evidence of early tubercle. Sometimes recurrent pyrexia results from sinus infection.

In allergic conditions, such as asthma, it is usual to find that the patient has previously suffered from infantile eczema, urticaria, hay fever, or some similar manifestation.

In cases of recurrent bronchitis it is necessary to inquire about the condition of the upper respiratory tract, for a history of repeated colds or tonsillitis suggests that the infection is in reality descending to the bronchi from some focus in the upper part of the respiratory tract.

Attention must always be paid to a *history of contact* with infection. This is of particular importance in the case of contact with open tuberculosis. It is also helpful in children with apparent bronchitis who have been in recent contact with measles or whooping-cough. A *chronic infective condition* in a patient who is connected with cattle should suggest the possibility of actinomycosis.

Finally, it is desirable, in obscure cases, to consider the possibility of conditions which do not commonly occur in this country, such as *amoebiasis*, *fluke infections*, and *parasitic cysts*. To this end inquiry should be made whether the patient has resided abroad.

### The Family History

Consideration of the family history is chiefly of importance in that it directs attention to hereditary predispositions, and that it indicates the likelihood of contact with infection at some stage in the patient's life.

The most definite familial respiratory affection is *allergic asthma*, in which cases a family history of that complaint, or of allied phenomena, is frequently noted. In certain families there is a tendency to the development of emphysema and chronic bronchitis. *Catarrh* tends to run in families.

In suspicious cases a family history of *pulmonary tuberculosis* may be of considerable value in suggesting the diagnosis in the early stages of the disease, but it is also of importance in forming a prognosis, for certain families appear to be unable to put up any effective resistance to the infection and, in such, the prognosis in the individual is below the average.

# SECTION II PHYSICAL EXAMINATION OF THE CHEST

## CHAPTER 5 INSPECTION

THE careful performance of the routine physical examination will furnish facts which, taken in conjunction with the information previously elicited in the history, will usually form the basis for a sound clinical diagnosis when there is moderately extensive disease. On the other hand, it cannot be too strongly emphasized that there may be no physical signs of any sort, and yet disease may be present. Therefore it must be clearly understood that, *whereas positive physical signs mean that there is some abnormality in the respiratory tract, the failure to elicit physical signs does not mean that the chest is healthy.* Although this fact has been obvious for many years, it is being constantly stressed by the increasing use of X-rays in diagnosis, and no physical examination can now be considered to be complete without an X-ray film. For this reason chest radiology has now been included in this section on the physical examination of the chest.

Physical examination must invariably be performed in the following order :—

Inspection.

Palpation.

Percussion.

Auscultation.

X-ray examination.

### Inspection

Inspection of the chest is the first, and a most important, method of physical examination, and in no circumstances may it ever be omitted. Attention should be directed, in this order, to the following points :—

General observations.  
Shape of the chest.  
Respiratory movements.

The cardiac impulse.  
Additional observations.



*General Observations*

It is a wise maxim to observe the patient before devoting attention to any particular system. It is this preliminary summing-up which is the hall-mark of the good clinician, and it is an art which cannot be explained in print, for the ability to "size up" a patient can only be acquired by experience. Yet this faculty is an essential feature which in reality forms the basis of clinical insight, or apparent intuition, and it can only be learned by painstaking observation of mankind in general, and not only of the sick.

At the very first glance it is usually possible to surmise whether the patient is well or ill and, if so, how ill. Appearances are often deceptive and mistakes are bound to be made, but increasing experience will be found materially to reduce the number of errors of judgement. The most obvious and striking general change is the presence of *cachexia*; this term implies more than simple wasting, although it is not easy to define the difference. The cachectic patient looks ill, the skin is atrophic and shiny, and one has the impression of impending death; advanced *carcinoma* or *tuberculosis* cause cachexia which, for contrast, might be compared with the simple wasting of a severe case of toxic goitre.

The absence or presence of *respiratory distress* is noted almost subconsciously; the causes have been mentioned in the section on dyspnoea. It is important not to overlook the presence of Cheyne-Stokes breathing.

The complexion may reveal much to the observant. There is the delicate texture of the skin and the "pink and white" appearance of tuberculosis, often associated in the active stages with a hectic flush, not to be confused with the malar flush of mitral stenosis. Many sufferers from chronic bronchitis and asthma have a rather bloated appearance from venous congestion, and a sallow or muddy hue is not uncommon when there is chronic suppuration. Rarely the skin will show a waxy appearance suggestive of amyloid disease.

Cyanosis is often seen, and it may be noticed earliest in the lips and ears. To understand its significance it is necessary to know how it is caused. Cyanosis results from an excessive amount of reduced haemoglobin in the capillary circulation, and the main factors concerned are *venous stasis*, *increased tissue*

*consumption of oxygen, deficient saturation of arterial blood, and a "shunt" from the venous to the arterial side.*

So far as respiratory disease is concerned cyanosis is a usual feature of *respiratory obstruction, pneumonia, and emphysema*, but there is often a concurrent cardiac factor. Acute respiratory obstruction due to any form of strangulation, oedema of the larynx, or large foreign body is a fair example of arterial anoxaemia; spontaneous pneumothorax and massive collapse of a lung sometimes produce a similar result. The cyanosis of pneumonia is more complex. There is increased tissue consumption of oxygen and, in many cases, a degree of capillary stagnation, but the "shunt" is also operative in the early stages; although a lobe is solid and airless the flow of blood through it is not impeded at first, so that a certain proportion of unacrated blood finds its way into the arterial circulation. The cyanosis of emphysema is chiefly due to stagnation of the circulation through the lungs and in the tissue capillaries, the arterial blood being well aerated and the venous blood more desaturated than normal.

Localized oedema and distended veins are a sign of blockage of the venous return and often signify a growth. From a consideration of the venous drainage of the parts concerned it is not usually difficult to locate the site of the obstruction.

### *The Shape of the Chest*

In bygone days it was customary to make an actual tracing of the outline of the chest by means of an instrument known as the cyrtometer. This is now rarely done, and it should be sufficient to obtain a general impression from inspection. For this purpose the examiner should stand facing the patient and looking towards the middle line of the chest. A satisfactory view can be obtained by standing at the foot of the bed, when the whole of the front of the chest can be taken in at a glance. It may be said that this is *not* a satisfactory method, as only one side is being looked at. This is true, but the chest is somewhat conical, and when studying the symmetry of the thorax is somewhat overlooked. When studying the symmetry of the thorax it is necessary only to note facts and to postpone deductions until inspection has been completed.

In the first place there may be *flattening of one side of the chest*. This usually indicates disease in the lung on the affected

*side, either fibrosis or collapse.* An appearance of flattening is rarely produced by wasting of the chest muscles, as in poliomyelitis or when the *pectoralis major* is congenitally absent. The spine should always be examined to exclude the presence of scoliosis.

Alternatively, one side of the chest may be *expanded*. This is apt to lead to confusion, for it is obvious that the *general* shape of the chest will be the same as when the opposite side is flattened. It is therefore always necessary to be very sure which side is sound and which is diseased, otherwise serious diagnostic error will result. Expansion nearly always indicates disease in the *pleura* on the affected side, the common causes being *pleural effusion* and *pneumothorax*. Very rarely a rapidly growing tumour may produce a similar result.

### *The Respiratory Movements*

The preceding points are noted while the general appearance of the chest is being observed. It is now necessary to pay closer attention to the act of breathing, as shown by the respiratory movements. The patient should first be allowed to breathe naturally. Normal breathing should be quiet and effortless; the rate should be about 20 per minute and the rhythm should be regular. Increase in rate is particularly marked in *lobar pneumonia* and this point is of great importance in the distinction between pneumonia and pneumonitis (p. 104). The presence of wheeze, stridor, or other abnormality may be observed and the action of the accessory muscles of respiration should be noted. In an acutely ill patient there may be dyspnoea, hyperpnoea, or Cheyne-Stokes breathing, the significance of which have been discussed. There is often a *characteristic expiratory grunt in pneumonia*; in the bronchopneumonia of small children the respiratory rhythm may be reversed, the sequence being (i) expiration, (ii) inspiration, (iii) pause, and this observation is therefore diagnostically significant.

When there is no dyspnoea it is desirable to observe the patient when he takes a deep breath, as this may accentuate any asymmetry of movement.

(a) *The respiratory excursion may be diminished.* This is most commonly seen in *emphysema*, because the chest wall is in a state of more or less permanent expansion; in case of

doubt the chest expansion should be measured. The minimum expansion in a healthy adult should be at least two inches.

(b) *Asymmetry of movement.*—This indicates that there is unilateral disease, or that the damage is appreciably greater on one side. In such cases *the affected side is the one which moves less well.* An appreciation of this clinical point is the secret of the successful interpretation of physical signs and, when taken in conjunction with the position of the mediastinum, it affords an indication of the type of lesion which is present.

### *The Position of the Cardiac Impulse*

It is now necessary to note the position of the heart, as this gives valuable information about the position of the mediastinum. The cardiac impulse is looked for in its usual situation, about three and a half inches from the middle line in the fifth left space. In a normal individual it should be easily visible but, in certain circumstances, some alteration may be noted.

(a) *The cardiac impulse may be invisible.* This may result from a variety of causes.

1. It is rarely visible in the obese.

2. *Pericardial conditions.* with fluid in the pericardium the heart is separated from the chest wall and the impulse is not seen.

3. *Myocardial conditions:* in failure the heart muscle may act so poorly as not to cause any visible impulse. This may also occur in conditions associated with malnutrition and with very low blood pressure.

4. *Pulmonary conditions:* in the normal person the anterior surface of the heart is to a large extent uncovered by lung and the normal cardiac impulse depends upon this. If for any reason the lung becomes more bulky, as it commonly does in *emphysema*, the heart may be displaced from its contact with the chest wall and the cardiac impulse may disappear.

5. *Pleural conditions:* the left pleural sac extends over part of the front of the heart in advance of the lung margin, and the development of *effusion*, or the occurrence of *pneumothorax*, in the left pleural cavity may have the effect of separating the heart from the chest wall.

It follows therefore that the fact of the disappearance of the cardiac impulse may be of considerable significance and it should always be carefully noted.

(b) *The cardiac impulse may be displaced to the left.* There are three sets of circumstances in which this may occur.

1. *The heart may be enlarged to the left.* This may result from dilatation of the ventricles in heart-failure: it is only seen in patients who are acutely ill. The impulse is feeble and wavy, the heart's

action is rapid, and the clinical state of the patient is that of heart-failure.

Enlargement to the left may also occur from *hypertrophy of the left ventricle*. The common causes are *high blood pressure, aortic regurgitation, and adherent pericardium*. The hypertrophied ventricle causes a pronounced impulse which may be seen over a wider area than usual and is well sustained.

2. *The heart may be drawn to the left.* This occurs when the volume of the left lung is diminished. The pleural space is potential, and not actual, so that the shrinkage of the lung must be compensated by movement of the heart and other mediastinal structures towards the affected side. The two common causes of traction to the left will therefore be *fibrosis and collapse of the left lung*.

3. *The heart may be pushed over to the left.* This almost invariably results from conditions affecting the *right pleural sac*, and the two common causes are *pleural effusion and pneumothorax*.

(c) *The heart may be displaced to the right.* The only primary cardiac condition in which this occurs is *dextrocardia* in which, owing to maldevelopment, the position of the heart is reversed in the chest. The heart is *drawn* to the right by fibrosis or by collapse of the *right lung*, and it is pushed over to the right by *left-sided pneumothorax or pleural effusion*.

The information obtained from the observation of the respiratory movements and of the position of the heart may now be combined and an idea may be formed of the type of lesion likely to be present.

Bearing in mind that the side which moves less well is the damaged side, it need only be stated that—

(i) Displacement of the mediastinum *towards the immobile (i.e. damaged) side results from fibrosis or collapse of the lung*.

(ii) Displacement of the mediastinum *away from the immobile (i.e. damaged) side results from pleural effusion or pneumothorax*.

*Additional observations.*—Campbell de Morgan's Spots (p. 24) are often seen in middle-aged patients; they have no significance, but care must be taken not to confuse them with ■ purpuric rash. Nodules of metastatic growth are sometimes observed in or under the skin. These vary in size and they may be quite large; it is obviously important that their significance should be recognized. Distended veins over the front of the chest indicate *venous obstruction* and they may therefore be an early sign of mediastinal tumour.

*Abnormal pulsation* is occasionally seen in the second and third right spaces, close to the sternum, in cases of *aneurysm of the ascending arch of the aorta*. The pulsation is sometimes

difficult to detect unless specially looked for ; *the upper chest should be viewed with the eyes on a level with the sternum when aneurysm is suspected.*

It is always advisable to include the inspection of the upper part of the abdomen at this stage and to note the respiratory movement in this situation. This affords valuable evidence of the action of the diaphragm. A pronounced inspiratory prominence of the epigastrium shows that respiration is being carried on chiefly by means of the diaphragm, and is commonly seen in cases where the intercostal muscles are not performing their function ; it is present with emphysema. Inspiratory recession of the upper abdomen on one side only indicates that the phrenic nerve on that side is paralysed and is suggestive of malignant growth involving the nerve.

*Litten's sign* is of some value in distinguishing lesions above and below the diaphragm. When the patient is recumbent in a good light a shadow will be seen to move from above downwards in the lower part of the axilla during inspiration. This is due to the pull of the descending diaphragm causing recession of the intercostal spaces ; it indicates that the diaphragm is contracting and that it has normal contact with the chest wall. This moving shadow is not seen in *pleural effusion*, whereas it is present with subphrenic abscess.

In conclusion, the fingers must always be examined for the presence of *clubbing*. The fully developed stato, with bulbous finger-ends, is easy to recognize, but the earlier stages are more difficult. A slight degree of curving of the finger-nails and a fullness and shiny appearance at the base of the nails are suggestive of clubbing, but opinions sometimes differ as to what constitutes the early stage of the condition. Clubbing is found in most chronic chest diseases, *bronchiectasis*, *lung abscess*, *empyema*, and *growth* ; rarely is it present in uncomplicated tuberculosis. It is also found in congenital heart disease and in subacute bacterial endocarditis.

#### SUMMARY OF ESSENTIAL SIGNS ON INSPECTION

*Shape —*

*Symmetrical.*

*Flat*—tendency to bronchitis, tubercle, etc.

*Expanded*—emphysema.

*Asymmetrical.*

*Retraction*—fibrosis or collapse of the lung.

*Expansion*—pleural effusion or pneumothorax.

*Movement.*—*The side which moves less well is the diseased side.*



## CHAPTER 6

### PALPATION

THE first object of palpation is to confirm the observations already made on inspection. The respiratory movements may be more accurately determined by palpation and the apex beat may be exactly defined. Further information may, however, be obtained, and as a routine this method of examination should be continued as follows :—

1. The state of the muscles can be ascertained by gentle palpation, especially over the pectoral muscles. The tone of these is diminished in many wasting conditions, as well as in diseases which primarily affect the muscles themselves. At this stage of the examination tenderness may be elicited, which may be caused by local inflammation in the muscles or in their subjacent fibrous tissues, or may result from deep-seated inflammation affecting the pleura. Tenderness in the upper left chest is sometimes associated with degenerative lesions at the base of the aorta, or with disease of the coronary vessels. It is sometimes noticed, on tapping the muscles, that a twitch is elicited ; this is known as *myotatic irritability*, and it is found in many wasting conditions, especially in tuberculosis and carcinoma. A further stage of the same condition is seen when the muscle responds by forming a small transient lump at the point of impact, a phenomenon known as *myoidema*, which has much the same significance.

2. Any lumps or irregularities which have been noticed on inspection should now be carefully investigated in order to determine their consistency and anatomical relationships. *This part of the examination must always include palpation of the supraclavicular fossae and axillae for enlarged glands, and of the breasts for carcinoma.* Hard swellings in or under the skin, especially when they are adherent to surrounding structures, nearly always indicate malignant growth. Fluctuation will be elicited when pus is present, either from subcutaneous supuration, abscess in connection with the spine or with a rib, or the rare cases in which an underlying empyema comes to the surface. It may also be present over a haematoma. All

swellings should be examined for *expansile pulsation*, a sign which is conclusive of aneurysm. Rarely, a respiratory thrill may be felt. This may be produced either by pleural friction or by the presence of exudate in the larger tubes; these can be distinguished by auscultation, especially if it be found that the thrill is altered or abolished by coughing. The crackling of air in the subcutaneous tissues (surgical emphysema) is very characteristic.

3. It is next necessary to determine the position of the mediastinum. This is done, firstly, by estimating the position of the heart. The *apex beat* is defined as that point furthest downwards and outwards at which the cardiac impulse can be distinctly felt, and normally it is situated three and a half inches from the middle line in the fifth left intercostal space. The causes of change in the position of the cardiac impulse have already been considered, but the great value of location of the apex beat lies in the fact that it affords a measurable index of displacement. Secondly, the position of the trachea can be determined by pressing gently backwards above the jugular notch. Normally this structure should lie exactly in the middle line, but if it should deviate, the finger will encounter lessened resistance and the trachea will then be found to be on one side. This displacement occurs for exactly the same reasons as displacement of the heart, and the two structures are often displaced together; therefore the trachea is displaced towards the affected side in fibrosis or collapse of the lung and away from that side in pleural effusion and pneumothorax.

4. The vocal vibrations are next elicited. This is done by placing the flat of the hand on the chest and instructing the patient to say "ninety-nine". This particular numeral is selected because it brings out the resonance of the voice in the most satisfactory way. The two sides of the chest must not be examined simultaneously, but corresponding areas must be compared consecutively, using the same hand.

There is no normal standard of vocal vibration. The response elicited varies according to the thickness of the chest wall and the depth of the voice. The vibrations are best carried by a thin chest but they are naturally weaker with a high-pitched voice than with one of low pitch. Due allowance should be made for these facts in assessing the result of this test. So long as the response is equal on the two sides no



definite conclusion can be drawn. *Increase* in the vocal vibrations is found over areas of pneumonic consolidation, and this sign is useful in distinguishing between consolidation and effusion. Occasionally an increase is noted over cavities in the lung. It is *more common* to find diminution. In women and children and in the obese the vocal vibrations are normally feeble and, of course, vibrations are absent when the voice is lost. Thickened pleura, bronchial obstruction, fibrosis of the lung, dense consolidation, tumour, and pneumothorax may all cause diminution, and *the vocal vibrations are commonly absent over pleural effusions*. Absent vocal vibrations are, in fact, a valuable indication of the presence of fluid in the pleura. In cases of pneumonia the vibrations are sometimes found to disappear rather suddenly; this event nearly always indicates a developing pleural effusion, and it may be a warning of impending empyema.

When there is both fluid and air in the pleural cavity a *succussion splash* may be felt. This is elicited by placing the flat of the hand on the back of the chest and moving the patient slowly forwards, and sharply backwards; it is pathognomonic of *hydro-pneumothorax*.

Finally, the upper abdomen must be palpated, for information may be obtained which is strictly relevant to the state of the respiratory tract. The liver or spleen may be found to be displaced downwards by a pleural effusion, or the liver may be enlarged from the presence of metastases or of amyloid disease. Sometimes a tumour may be felt in the abdomen which gives rise for the first time to the suspicion that the trouble in the chest may be due to secondary deposits from an extra-thoracic source.

#### SUMMARY OF ESSENTIAL SIGNS ON PALPATION

##### *Position of Mediastinum.*

*Displaced towards the affected side*—fibrosis or collapse of the lung.

*Displaced away from the affected side*—pleural effusion or pneumothorax.

##### *Vocal vibrations.*

*Increased*—consolidation of the lung.

*Diminished*—most chronic lung conditions, thickened pleura.

*Absent*—pleural effusion.

## CHAPTER 7

### PERCUSSION

It is desirable to acquire a reliable technique in percussion at the earliest possible stage of the clinical course. The act requires much practice and, although percussion is very variously carried out, the results are equally reliable so long as the observer always uses the same method. The chief points which make for a good technique are as follow :—

1. A finger, usually the left middle finger, is placed firmly on the chest wall. As little of the finger as possible, preferably only the terminal phalanx, is used and it is held firmly in contact with the skin.
2. The finger should be placed either along the axis of the intercostal space or upon the rib.
3. The finger which gives the percussion stroke, the "plessor" finger, which is the index or middle finger of the right hand, should be brought down sharply at a right angle, and should be withdrawn immediately. If allowed to rest on the "pleximeter" finger, the percussion sound is modified.
4. Comparable areas of the chest wall should be examined consecutively as a rule; spaces must always be compared with spaces and ribs with ribs. At a later stage of the examination, when areas of impaired resonance are being mapped out, or when the area of cardiac dullness is being investigated, it is the rule to percuss *from the more resonant to the less resonant area*.

The mechanism of production of the percussion note is complex and it is not necessary to consider it in detail. It is sufficient to observe that the note can be resolved into three main elements, and that, in addition, valuable information may be gained from the sense of resistance to the percussion stroke.

The loudness of the note varies according to the force employed in striking and it is not important.

The longer the duration, the more full and resonant the note. The *pitch* is a more definite, and more important, attribute. Low-pitched tones are called "tympanitic", the note produced by percussing the gas-distended abdomen. A slight rise in the pitch, and a note more commonly elicited over the lungs, is termed "sub-

tympanitic". As the quantity of air in the percussed area diminishes, so the pitch of the note rises, but special names need not be attached to the different sounds. The pitch varies according to the tension of the contained air; the higher the tension the higher the pitch, and the less the resonance.

The tone of the percussion note is seldom pure. Mostly it is muffled and, with complete dullness, there may be sound without tone.

The *sense of resistance* felt on percussion is, to the expert, almost as significant as the sound produced. The sensation with fluid is one of unyielding resistance, which is characteristic. With all but the most dense consolidation there is some sensation of elasticity even although there be little resonance.

It is customary to describe the percussion note in abstract terms according to its "resonance". The term is really used to indicate the result of a subconscious mental combination of the pitch and duration of the sound, and the note is described as being more or less resonant than normal, or as being dull. The ability to describe and to interpret the percussion note must be acquired by experience.

The objects of percussion are, firstly, to elicit a sound and, secondly, to determine the sense of resistance. The physical state which is examined is the depth of the column of air which underlies the percussed part. The longer the column, and the less the amount of overlying tissue, the more resonant the note.

There is no normal percussion note. It varies with the thickness of the chest wall and with the vigour of the percussion stroke. It is best to adopt a stroke of medium intensity as a standard. A stroke which is too heavy will elicit resonance from areas far removed from that which is being percussed, whereas a stroke which is too light will fail to elicit resonance which is subjacent. The note may vary in either of two ways, being more or less resonant than the normal, which is judged by the character of the note on the apparently sound side of the chest.

A more resonant note is found when there is more air in proportion to solid tissue. It is present in *emphysema* and, as that condition is usually bilateral, it may not be possible to be sure of the change until the condition is advanced. A hyper-resonant note is obtained over a *pneumothorax*. In most cases the note is low in pitch and of markedly increased resonance, although this is not always the case, especially when the lesion

is localized; the pitch varies with the tension of air in the cavity, becoming higher as the tension increases. When the lung tissue is relaxed, as occurs in the lobe situated above a pleural effusion, a tympanitic note may be elicited which is characteristic and to which the term "Skodaic" resonance is applied. In bronchopneumonia in children, especially if percussion is carried out when the child is crying, a high-pitched, yet resonant, note is sometimes heard ("cracked-pot" sound).

An impaired note may result from causes in the lung or in the pleura. Lung conditions which give rise to impairment of note are those in which the proportion of solid tissue to air is greater than normal, and any consolidation of sufficient size will produce this effect. It is present in most cases of pneumonia, tuberculosis, and growth; also with collections of fluid within the lung, such as lung abscess and hydatid cyst. Collapse of the lung results in considerable impairment of note over the airless part, and fibrosis has a similar effect. Should the latter condition be bilateral it may be difficult to obtain much information from percussion unless the change is advanced. Thickening of the pleura causes impairment of note, but the thickening must be considerable before any marked change is noted; in most cases the impairment is due at least as much to disease in the underlying lung as to change in the pleura. Pleural effusion is a very common cause of gross impairment, and there is usually complete absence of resonance, the only condition, in fact, in which the term "dull" can strictly be applied.

It must be kept in mind that small areas of consolidation which are surrounded by air-containing lung may yield no detectable change in the note; also that in cases of chronic shrinkage of lung tissue, such as happens with localized fibrosis or collapse, compensatory emphysema is likely to occur and may balance the diminished resonance, so that the note may appear normal. So long as it is remembered that percussion merely determines the amount of air in the part, serious error should be avoided.

Having noted the general percussion characteristics, it is next necessary to pay attention to matters of detail. In the first place, the shape of any area of impairment must be mapped out. The areas which result from tuberculosis and from growths are usually indefinite, in striking contrast to the exact lines of demarcation, adhering strictly to the anatomical limits of t

affected lobe, which may be demonstrable in lobar pneumonia. In pleural effusion of moderate degree the shape of the dull area is very characteristic, for the upper border rises from the region of the spine with a convex upper margin to reach a peak about the mid-axilla, and gradually slopes down towards the sternum. In doubtful cases the shape of the dull area may be of considerable diagnostic help. The dullness of a localized collection of fluid, such as an interlobar empyema, an aneurysm, or a cyst, may be very sharply defined, as may be that of some solid tumours.

Finally, the area of cardiac dullness should be mapped out. From the point of view of the examination of the respiratory system this is important chiefly in that it checks the observations on the position of the heart, and affords useful information as to whether the heart is enlarged or displaced. Sometimes, as in cases of pericardial effusion, the area of cardiac dullness may encroach to a considerable extent upon the area normally occupied by the lung on either side, and difficulty in diagnosis may result. Pericardial and left-sided pleural effusions are occasionally confused, although careful attention to the shape of the dull area should obviate error as a rule.

#### SUMMARY OF ESSENTIAL SIGNS ON PERCUSSION

##### *Percussion note.*

*More resonant*—emphysema, pneumothorax.

*Less resonant*—any condition in which the lung contains less air, consolidation, collapse, fibrosis, thickened pleura.

*Dull*—pleural effusion, massive growth.

## CHAPTER 8

### AUSCULTATION

The stethoscope is the conventional sign of the doctor, and it has so far been his main instrument of precision in the diagnosis of diseases of the heart and lungs. It is therefore obvious that a good stethoscope is essential, and yet most students are prepared to accept whatever is offered to them. In some cases the stethoscope supplied is practically a non-conductor of sound, and this accounts to a large extent for the lack of progress commonly made by the junior student. The selection of a proper stethoscope should always be carefully carried out and advice should be taken before a final choice is made. It matters not what type is chosen so long as one is sure that the best possible conduction of sound is obtained.

In order to appreciate the significance of sounds heard through the stethoscope it is necessary to listen with a definite object in mind, and to evaluate the various sounds heard in a definite order. The beginner will find it helpful to auscultate with a view to being able to answer the following questions:—

1. Are breath sounds present, and if so, are they of equal intensity on the two sides?
2. What is the quality of the breath sounds?
3. Are added sounds present, and if so, what is their nature?
4. What is the nature of the voice sounds?
5. Certain additional observations.

The intensity of the breath sounds is a guide to the state of the underlying lung. It is best first to listen while the patient is breathing naturally, and again while deep breaths are being taken. The sounds may be more intense than normal whenever there is over-breathing. This may occur in the sound part of the lung when an extensive area is airless for any reason. The common abnormality is, however, weakening of the breath sounds. It is an axiom that the less intense breath sounds are present over the damaged area, and this diminution may result from any kind of disease, either in the lung (consolidation, fibrosis, collapse) or in the pleura (thickened pleura, effusion,

pneumothorax). The intensity of breath sound, therefore, runs roughly parallel to the respiratory movement. The sounds will be equally diminished on both sides of the chest when both lungs are equally damaged, as occurs in emphysema or diffuse fibrosis.

The breath sounds are entirely absent when, for any reason, air is unable to enter a large bronchus. Obstruction of a bronchus by *foreign body* or by *growth*, leading to *massive collapse* of the lung, or pressure on the lung from a large *pleural effusion* or *pneumothorax*, are the most likely causes.

The *quality* of the normal breath sounds is termed *vesicular*. The column of air is conducted along the trachea and main bronchi in a steady stream, and it is then broken up in the innumerable pulmonary alveoli, so that the pure sound which is present over the main tubes is altered. It is customary to compare vesicular breathing with the sound produced by the rustling of leaves in the wind, yet this comparison is not really adequate, and the normal vesicular breath sounds can only be fixed in the mind by careful study of the normal chest. Vesicular breath sounds are normally heard over the whole of the lungs with the exception of a small area in the second right space, close to the sternum, where the eparterial bronchus approaches the surface.

It is helpful to remember that, with vesicular breathing, the sound is audible throughout inspiration, there is no perceptible pause between inspiration and expiration, and the sound is only heard in the first quarter of expiration, being less audible in this phase. Loud vesicular breathing is often heard in the chests of children and is termed *puerile*. *Prolongation of expiration* is often the earliest evidence of disease. It is commonly heard when emphysema is present and, when it is localized to an apex, it may be a sign of early tuberculosis. An irregular breath sound, *cog-wheel breathing*, may be heard, chiefly over the upper lobes and anterior margins of the lungs. It is due to irregular expansion of the lung, sometimes as a result of uneven descent of the diaphragm, but its significance is doubtful; it need not necessarily mean disease in the lung or pleura.

Abnormal breath sounds are frequently heard, the common variety being known as *bronchial breathing*. In this the quality of the breath sounds is *pure*, inspiration and expiration are of

## AUSCULTATION

47

approximately equal length, and there is a pause between the two phases of the cycle. Occasionally the breath sound has the characters both of vesicular and of bronchial breathing, and to this the term "*broncho-vesicular*" is applied; the significance of this sound is very varied.

True bronchial breathing occurs when the pure sound produced in the bronchial tubes is conducted without alteration to the stethoscope. The conditions which are necessary for its production are: firstly, that the *bronchus must be patent*; and secondly, that there must be *increased homogeneity of the lung tissue*. The lung parenchyma may be compared with a sponge, in that there is a solid network containing enmeshed air spaces. Any change which causes an increase in either element at the expense of the other will therefore tend to produce bronchial breathing. Destruction of lung tissue will result in an increase in the contained air, and consolidation will have the reverse effect, but in either case the tissue will be more homogeneous. It follows that *cavitation or consolidation* may equally be the cause of bronchial breathing, provided always that the bronchus leading to the damaged area is patent. It is not always realized that a small *pleural effusion*, which is not large enough to shut the lower bronchus, will produce this same physical state and that therefore bronchial breathing may be heard in the earlier stages of this condition, for fluid is a good conductor of sound. The more intense varieties of bronchial breathing are sometimes termed "*tubular*", but this term has no special significance. When the breath sounds are very intense, and when an echoing or metallic quality is present in addition, the term "*amphoric*" is used; this type of sound is heard over large cavities, and in some cases of pneumothorax. Added sounds are of several varieties. They may be classified as follows:—

- (a) Continuous.  
*Rhonchi and sibili.*
- (b) Interrupted.  
*Rales.*
- (c) Crepitations.  
*Extra-pulmonary added sounds.*

*Rhonchi and sibili* are produced by a narrowing of the bronchial tubes which may result either from catarrhal exudation or from spasm. Thus they commonly indicate the presence



of *bronchitis* or of *spasmodic asthma*. The *rhonchus*, a low-pitched sound, is produced in the larger tubes, and the *sibilus*, high-pitched, is produced in the bronchioles.

*Rales* are produced by the bubbling of air through liquid, and they are minute explosive noises which usually occur in showers. They vary in size and may be termed coarse, medium, or fine, according to the size of the tube in which they are produced. *Rales* are commonly heard in the region of cavities and usually indicate the presence of *tuberculosis*, *bronchiectasis*, or *lung abscess*, but they are also heard when there is exudation of fluid into the bronchioles, as occurs in *congestive heart-failure* and *oedema of the lungs*.

*Crepitations* are the finest of all added sounds and they are heard only in inspiration. It may be difficult or impossible to distinguish a crepitation from a fine rale. The crepitation is produced by undue adhesion of the alveolar walls on expiration, so that the inspiratory effort forces them apart, producing an audible sound in the process. *The presence of crepitations, therefore, is a definite indication of damage in the lung tissue itself.* Crepitations are commonly heard in the early stages of *pneumonia* ("indux"), as well as during resolution ("redux"), in early *tuberculosis* before cavitation has occurred, and in cases of incomplete collapse of the lung ("atelectatic" crepitations). They are sometimes heard at the beginning of auscultation in debilitated subjects as a result of patchy collapse of small areas of the lung. Such crepitations may be recognized by the fact that they disappear on deep inspiration or after coughing, when the lung is caused to expand better.

*Extra-pulmonary sounds* may be of many types, from the coarse audible tremor which is sometimes produced in muscle to the crackling noises which may be heard in *surgical emphysema*. (The nature of this latter sound will be made clear on palpation, for the crackling can be felt as well as heard.) The only added sound which has any significance so far as the respiratory tract is concerned is *pleural friction*. The sound is usually coarse and creaking in character, although sometimes it is fine and may resemble crepitation. Friction is localized and unilateral, it is generally heard in inspiration and in expiration, it is not altered by coughing, but the sound may be increased by pressure of the stethoscope on the affected area. Pain and tenderness are usually present where friction is heard.

After the added sounds have been determined the patient is instructed to cough, and any alterations are noted. Vanishing sounds are rarely significant, but rales and crepitations which are accentuated by coughing are sometimes a sign of early tubercle.

*Post-tussic suction* consists in a curious sucking noise, heard after coughing, which is caused by air rushing into a cavity. It is said to be the only sign which indicates a cavity with certainty, but it is very rarely heard.

The *vocal resonance* is studied when the patient says "one, two" or "ninety-nine"; both are suitable. The normal voice sounds are indistinct, and in this respect they are comparable with vesicular breathing, for the clear laryngeal tones are lost when the air column is split up in the alveoli. Experience is all that is necessary to be able to recognize normal voice sounds. The resonance is faint when the voice is high-pitched, when the patient is well-covered, and when the voice itself is weak; it is strong when the reverse conditions are present.

*Bronchophony* is the commonest abnormality. It occurs when the pure voice sounds are conducted without change to the stethoscope, and it may be defined as "the *clearest* degree of vocal resonance ever heard over the healthy chest, and all degrees more clear than this". The loudness of the sounds is nothing to do with bronchophony; it is simply a matter of *clearness*. When in doubt, the sounds may be compared with those heard in the second right space, close to the sternum, where normally the *clearest* vocal sound is heard; equal or increased clearness constitutes bronchophony. The causation of bronchophony, direct conduction from the bronchus to the stethoscope, is precisely similar to that of bronchial breathing. These two phenomena will therefore be found in similar circumstances, in brief, *consolidation* and *cavitation*, and the two conditions cannot be distinguished with certainty by the voice sounds.

*Aegophony* is a variety of bronchophony, but the clear sounds are altered by the addition of a nasal element; it owes its name to its fancied resemblance to the bleating of a goat. Although it may occur in any of the conditions which cause bronchophony, it is most commonly heard over a thin layer of fluid in the pleura.

*Whispering pectoriloquy* is a term employed to indicate the direct transmission of syllables by the whispered voice. It is

heard over cavities which are in direct communication with a bronchus, and sometimes over solid areas. This sign may be helpful in the case of very ill patients or when the voice is weak.

Additional observations may be made during auscultation. Should there be reason to suspect the presence of a *pneumo-thorax*, an attempt should be made to elicit the *coin sound*. This is done by placing a coin flat on the chest and percussing it with another coin while auscultating. Lung tissue produces a dull note, whereas a large air space yields a clear, echoing sound. The sound is not present over every pneumothorax, for the tone depends on the tension of air within the cavity, and it may happen that the result varies from time to time in the same patient. A positive coin sound is almost diagnostic of pneumo-thorax, although it may be elicited, very rarely, over enormous cavities in the lung.

A physical sign which is pathognomonic of hydropneumo-thorax is the *succussion splash*. This occurs when there is a moderate quantity of fluid and a good deal of air in the pleural cavity. It is best elicited by listening at the suspected upper level of the fluid and jerking the patient slowly forwards and sharply backwards. A splash is heard which is quite characteristic, and the impulse may even be perceptible to the patient and to the hand of the examiner. When there is only a small quantity of fluid there will be no splash, but a *metallic tinkle* may be heard on deep inspiration. This is a musical sound which somewhat resembles the bursting of a bubble. It often accompanies amphoric breath sounds and is said to be due to the bursting of bubbles which ascend through the fluid from a tiny opening in the lung below the level of the liquid.

The physical examination of the chest completed, a careful summary of the physical signs should be prepared and an attempt should be made to interpret them in the light of the history. It must not be forgotten that a complete examination of the patient must be made in every case, for fresh information may be gained which may confirm the opinion already formed or which may, on the other hand, suggest other possibilities. In either case the clinical diagnosis is rarely sufficient, for it is usually capable of proof, and, with this end in view, the appropriate methods of special investigation should next be applied.

## SUMMARY OF ESSENTIAL SIGNS ON AUSCULTATION

*Breath sounds.**Absent*—bronchial obstruction, pneumothorax, effusion.*Weak*—emphysema, bad breathing habit, thick chest wall, fibrosis, partial collapse, small effusion.*Bronchial*—consolidation or cavity, *provided that the bronchus is patent.**Added sounds.**Rhonchi and Sibilli*—bronchitis, asthma.*Rales*—air bubbling through liquid. Tuberculosis, bronchiectasis, abscess, congestion or oedema of lung.*Crepitations*—lesion of the lung parenchyma—pneumonia (early and late), tuberculosis, incomplete collapse.*Voice sounds.**Bronchophony*—as for bronchial breathing.*Egophony*—thin layer of fluid in the pleura.

## SUMMARY OF ESSENTIAL PHYSICAL SIGNS

(Abnormalities refer to the affected side)

|                       | Collapse   | Fibrosis                                   | Consolidation               | Effusion                 | Pneumothorax              |
|-----------------------|------------|--|-----------------------------|--------------------------|---------------------------|
| <b>INSPECTION :</b>   |            |  |                             |                          |                           |
| Asymmetry . . .       | Flattened  | Flattened                                  | None                        | Expanded                 | Expanded                  |
| Movement . . .        | Diminished | Diminished                                 | Diminished                  | Diminished               | Diminished                |
| <b>PALPATION :</b>    |            |  |                             |                          |                           |
| Mediastinum . .       | Towards    | Towards                                    | Unchanged                   | Away                     | Away                      |
| Vocal vibrations      | Less       | Vary                                       | Increased                   | Absent                   | Vary                      |
| <b>PERCUSSION :</b>   |            |  |                             |                          |                           |
| Note . . . . .        | Impaired   | Impaired                                   | Impaired                    | Dull                     | Resonant                  |
| <b>AUSCULTATION :</b> |            |  |                             |                          |                           |
| Breath sounds :       |            |  |                             |                          |                           |
| Intensity . . .       | Absent     | Weak                                       | Vary                        | Weak or absent           | Weak or absent            |
| Character . . .       | .          | Vesicular, (bronchial with bronchiectasis) | Bronchial                   | May be distant bronchial | May be distant "amphoric" |
| Added sounds          | None       | Rales with bronchiectasis                  | Crepitations early and late |                          | ..                        |
| Voice sounds . .      | Weak       | Weak                                       | Bronchophony                | Egophony                 | Vary                      |
| Special signs . .     | ..         | ..   | ..                          | .                        | Coast sound               |

## CHAPTER 9

### RADIOLOGY OF THE CHEST

THIS section deals with the general principles of the X-ray examination of the chest and the radiographic appearances of the common abnormalities. The best results are obtained by combining the information derived from the film with that gained by clinical observation. Physical examination of the chest is never complete until a good X-ray film has been carefully scrutinized. And, in fact, this method of examination will frequently reveal abnormalities which could not be detected by even the most competent observer who places his faith on physical signs alone.

The complete radiological examination consists in a routine preliminary screening of the chest and subsequent inspection of films taken in such positions as are considered necessary. Certain special measures may then be adopted in order to secure further information.

*Screening.*—Apart from conveying a general impression of the state of the lung fields, and this can be obtained in much greater detail from a study of the films, screening is essential in order to detect *abnormalities of movement*. The action of the diaphragm can be clearly seen, and the extent of its excursion. By this means accurate knowledge can be obtained concerning the mechanism of respiration; in the emphysematous subject the diaphragm is seen to lie lower than usual and to move little, if at all, on inspiration. Most asthmatics can be seen to make little use of the diaphragm and the lower part of the chest remains fixed. When one side of the diaphragm is paralysed, as it often is in cases of *bronchial carcinoma*, the affected side is seen to be considerably raised and either *immobile* or it may even ascend on inspiration (*paradoxical movement*). The pulsation of the heart and aorta should also be studied, although much of the information so obtained lies in the province of the cardiologist. The conus of the pulmonary artery can often be seen pulsating vigorously when there is any obstruction in the pulmonary circulation and the size of the aorta may be defined. It is necessary in all cases of apparent mediastinal tumour to

be certain that the shadow is not that of an aneurysm, and a simple test is to screen the mass in order to observe whether it pulsates.

*The film.*—The most satisfactory films are those taken with the tube six feet distant from the patient, so that the rays are parallel, or nearly so. A film should never be taken with the tube less than three feet from the chest, as an element of distortion is introduced and it is impossible to compare the size of the various structures with the normal. It is usual to take a *postero-anterior* film, that is, the tube is behind the patient and the cassette is held in front of the chest. This is the standard film which is always produced when a chest X-ray is asked for without qualification. It is sometimes found that a lesion is more clearly seen with an *antero-posterior* film; in this case the patient faces the tube while the cassette is held against the back.

*In addition to the standard postero-anterior film, a lateral view must always be taken when any lesion is present, in order to localize it* (figs. 23, 24). The necessity for a lateral view is particularly great in view of recent work which has demonstrated the existence of well-defined "segments" within the various lobes of the lungs. The demonstration of local segmental involvement is of great significance in the recognition of early malignant disease, and it has now become extremely helpful to the surgeon who is planning an operation for lung abscess or for bronchiectasis. It is not necessary for the student to be familiar with the names which are given to the many segments which have been described, nor, indeed, is the nomenclature fully established at present. Films are usually taken in the erect position and, if taken with the patient recumbent, allowance for this must be made when the film is examined. The patient must always be erect when it is desired to determine the presence of a *fluid level* (figs. 18, 23, 49). Care must be taken to ensure that the patient is straight before the exposure is made.

Abnormal appearances consist, firstly, in a divergence of the anatomical structures from the normal, and secondly, in the presence of abnormal shadows; it is in the interpretation of these shadows that difficulty is most apt to occur. A shadow is cast by any portion of lung which has become airless, and there is nothing specific about most of them. It is therefore unwise to draw too definite deductions about the nature of a disease

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from the examination of the film alone, and the X-ray examination must not be allowed entirely to supplant the methods of physical examination and the other investigations which have been described. Consolidation may be due to causes other than tuberculosis or growth, and cavities may be seen in many conditions besides tuberculosis.

The interpretation of X-ray films can be simplified if they are examined in a logical order, and the following routine is useful :—

1. *The bony chest wall.*—It is first necessary to be sure that the film is strictly postero-anterior, otherwise the structures will be distorted. This can be done by noting whether the trachea is situated centrally between the inner ends of the clavicles, so long as there is no cause of gross displacement of the mediastinum. When there is displacement it is necessary to note whether the inner ends of the clavicles lie equidistant from the central line, usually marked by the spinous process of a cervical vertebra.

The thoracic cage must next be inspected. In the normal chest (fig. 3) the ribs are seen to slant downwards and forwards from the transverse processes of the vertebrae, and the width of the intercostal spaces is between half and three-quarters of an inch in the adult. When the lungs are expanded, as occurs in emphysema, it is found that the ribs become more horizontal and the intercostal spaces are unduly wide. In thin subjects the ribs often slope more sharply downwards and the intercostal spaces are narrowed, and, when one lung is contracted, this change will be unilateral. At this stage of the examination

The arms should be so placed when the film is taken that the edges of the scapulae are withdrawn from the lung field. Otherwise the vertebral borders are seen as sharply defined lines running downwards on each side, but careful inspection should reveal their nature (fig. 5). It sometimes happens that the scapulae may cover a faint shadow in the underlying lung. It is possible in this way to overlook a small encysted collection of fluid, and great care must therefore be taken in reporting any film in which the bodies of the scapulae have been allowed to obscure part of the lung fields.

2. *The diaphragm.*—The appearance of the diaphragm should next be studied. Normally the right half is about an inch higher than the left and the surface should be smooth and well defined. Irregularities, "peaking" or "tenting", are produced by basal adhesions, and smooth projections sometimes indicate underlying tumour masses. The whole of the diaphragm may be lowered in position in cases where the lungs are expanded, as in *emphysema*; it may be raised when there is increased intra-abdominal pressure, as in obesity, pregnancy, ascites, or with large tumours, such as ovarian cysts. Asymmetry of the diaphragm results from developmental abnormality, from *paralysis*, or from subjacent collections of fluid, such as subphrenic abscess. Sometimes the diaphragm is seen as a very thin line high up in the chest; this occurs when the muscle has failed to develop, and the diaphragm is represented merely by a leaf of fibrous tissue, a condition known as *eventration* (fig. 8), which has little clinical significance. A further stage of this developmental abnormality is complete absence of the diaphragm on one side, with resultant diaphragmatic hernia (figs. 61, 62).

The paralysed diaphragm is higher than normal. Non-traumatic paralysis of the phrenic nerve is usually due to *malignant infiltration* (fig. 30). It is stated not to occur in cases of aneurysm and the observation is therefore useful in diagnosis. Paralysis of the diaphragm is frequently seen as the result of phrenic evulsion for the treatment of tuberculosis (figs. 59, 60).

In some cases, owing to dense opacity in the adjacent lung field, it is impossible to be sure of the exact position of the left diaphragm. In such cases the patient may be given a Seidlitz powder immediately before the film is taken, and the resultant gas bubble in the stomach outlines the position of the muscle.

It is only possible to clarify the outline of an obscure right diaphragm by inducing a pneumoperitoneum.

The area immediately above the diaphragm represents the base of the lower lobes. It is sometimes found that heavy breast shadows in middle-aged women obscure this area and render the film difficult to interpret. When it is thought necessary to obtain a good view of the bases in such a patient, a film can be taken while the breasts are raised by the patient. This procedure is of value in identifying the presence of shadows which would otherwise be overlooked. A similar result can be obtained by placing the patient in the prone position.

3. *The mediastinum.*—The position of the mediastinum is indicated by the position of the heart and trachea. It is important to have some idea of the size of the normal heart and not to confuse enlargement with displacement. A little experience is usually sufficient to ensure this. The significance of displacement of the mediastinum has already been fully indicated in the discussion on physical signs and it will be very briefly re-stated. The mediastinum may be drawn towards the affected side (as is determined by subsequent inspection of the lung fields) by *fibrosis* or *collapse* of the lung (figs. 13, 14). It is displaced away from the affected side by *pleural effusion* or *pneumothorax* (figs. 11, 12).

Although this rule is rather arbitrary, and many cases are encountered in which the presence of more than one condition simultaneously may complicate the radiological appearances, yet it forms a sound working basis and will enable the majority of skiagrams to be interpreted. When further information as to the position of the mediastinum is required, a barium swallow may be given and the position of the oesophagus can thus be defined.

The normal thymus does not cast a shadow, even in infants. Enlargement, especially increase in thickness, must be considerable before the gland can be identified.

Swellings in the mediastinum are often more obvious in the X-ray than on clinical examination. They may result from the presence of *abscess*, *enlarged glands*, *tumour*, *aneurysm*, or *cyst*.

(a) Abscess in the mediastinum is often ill-defined, and the diagnosis is made on the clinical state.

(b) Enlarged glands may result from *tubercle*, *lymphadenoma* or *growth*. Primary glandular tuberculosis is not

often seen in adults, although the glands are involved in most cases of primary tuberculosis in childhood. Deposits of calcium definitely indicate past tuberculosis (fig. 35). The glands of *lymphadenoma* form tumours of varying size but not usually very large. In the early stages there is a broadening of the aortic shadow, usually with a bulge to one side. Later a mass with wavy outlines may be seen projecting on each side (fig. 54); pulsation is not visible on the screen. The diagnosis can only be made for certain by the microscopic examination of a superficial gland. The glands may also enlarge in leukaemia.

(c) *Growths* may be primary or secondary. One type of bronchial carcinoma may infiltrate the mediastinum while not involving the lung fields (fig. 29). *Lymphosarcoma* forms a rapidly growing tumour; the rate of increase can be studied in serial films. It is very radio-sensitive, and the therapeutic test is of some value in differential diagnosis (figs. 57, 58). *Secondary masses in the mediastinal glands* may occur with carcinoma anywhere. Primary growths in the thymus are rare and tend to give a rectangular shadow. Very rarely fibroma, lipoma, and neurofibroma occur in the mediastinum. They all cause a well-defined, rounded opacity.

The thyroid gland may enlarge into the superior mediastinum, sometimes without obvious enlargement in the neck; it forms a pyramidal tumour with the base upwards (fig. 53). The aorta is displaced downwards and to the left and the trachea may be compressed. The shadow can be seen to move upwards on swallowing. These tumours are sometimes calcified.

*Aneurysm* may be very simple to recognize, for most tumours which show well-marked pulsation are of this nature. Considerable help may be obtained by a study of the kymogram (p. 69). The aorta may be dilated throughout a large part of its course (fusiform aneurysm) (fig. 55), or it may form a local bulge (saccular aneurysm) (fig. 56). When the swelling is small the connection with the aorta is usually obvious, but when it is large, it may be impossible to be sure of this. When there is much blood clot in the sac pulsation may not be seen and diagnosis may then be difficult. *Aneurysm does not paralyse the phrenic nerve*. In doubtful cases the clinical findings, Wassermann reaction, screen appearances, plain film, and kymogram must all be taken into consideration.

Cysts in the mediastinum are rare. *Dermoid cysts* arise in

connection with the thymus or the pericardium. They cast well-defined, round, or oval shadows, which are seen in the lateral view to lie in the *anterior mediastinum* (fig. 52). The presence of such structures as teeth makes the diagnosis obvious, but these are rare and the nature of the swelling is a matter of inference. *Hydatid cysts* are less common in the mediastinum than in the lung. There is nothing characteristic about them, but the walls sometimes show calcification.

4. *The lung fields.*—The appearance of the lung fields varies very greatly, according to the length of the exposure, the intensity of the rays, and the development of the film. For correct interpretation it is essential that the film should not be over-exposed, as much of the finer detail is lost and early disease may be missed. The normal lung fields are seen as translucent areas which are crossed by striate markings which radiate fan-like from the hilum on each side. These markings consist chiefly of the shadows cast by the blood vessels which accompany the bronchi and they are not caused by the bronchial walls themselves.

The fissure between the right upper and middle lobes may be visible as a thin sharp line, with an upward convexity, running transversely across the lung field in the fourth space. An accessory lobe is sometimes seen in the normal chest, the *azygos vein lobe* of Wrisberg (fig. 5). The azygos vein takes an unusual course across the right upper lobe and causes a deep indentation in its substance, the medial part forming the accessory lobe. The vein itself is not visible, but the pleural reflection is seen as a small triangular area in the upper part of the hilum, from which a thin sharp line curves obliquely upwards and outwards across the clavicle. Collapse, tubercle, and bronchiectasis are sometimes found in this abnormal lobe.

Alterations in the density of the lung give rise to two main types of picture.

(a) The lung may be more translucent than normal. This results commonly from *emphysema*, in which there is more air and less solid tissue than usual. In the common type of emphysema the change is universal, but sometimes, when a portion of the lung is collapsed, there may be compensatory emphysema which results in a local area of increased trans-

lucency. When this change is very gross it may sometimes be mistaken for pneumothorax, but the lung markings can be faintly seen in the translucent area (fig. 21).

Cavitation in the lung is the other cause of increased translucency. Cavities are nearly always surrounded by a well-defined wall, and the thicker the wall the more chronic is the cavity. It is sometimes difficult to be sure, when there is diffuse consolidation, if a cavity is present as well, and in such cases the presence of a fluid level is strong evidence in favour of cavitation. The tomogram may give valuable help in cases of this kind. When a fluid level is present in a large superficial cavity it is sometimes difficult to distinguish between an intrapulmonary cavity and a localized hydro- or pyo-pneumothorax. When the cavity is situated in the lung the fluid level never extends quite out to the chest wall whereas, with pleural effusion, the fluid level can usually be seen to reach the rib margins. Cavities may result from *tubercle*, *bronchiectasis*, *cyst*, *lung abscess*, or *breaking-down growth*. Recent and very active tuberculous lesions cause a thin-walled cavity which may be almost invisible on the film. In chronic phthisis the walls are thick and obvious (fig. 43). The presence of infiltration and calcification are confirmatory of tubercle. *Lung abscess* is usually well-defined, although the margins are often indefinite, and the presence of a fluid level is confirmatory evidence (fig. 23); breaking-down growth may be very similar in appearance. *Cysts* and *bronchiectatic cavities* have a thin wall which is sometimes difficult to identify (figs. 15, 17). These circles were at one time termed "pleural rings" but they have, in fact, no connection with the pleura.

(b) The lung fields may be less translucent than normal. This may result from the following changes:—

*Collapse* of the lung implies that the lung tissue has become absolutely or comparatively airless, and in such cases a shadow of varying density results. With complete collapse (fig. 13) the mediastinum is shifted towards the affected side, but if only one lobe collapses there may be little mediastinal displacement (figs. 21, 22) and the remaining lobe or lobes may enlarge in order to fill up the space. A collapsed lobe will therefore show a well-defined shadow which may be intensified by contrast with the translucency of the surrounding emphysema. The shadow caused by lobar collapse is triangular and shows a sharp

straight border. When the left lower lobe is collapsed, the shadow may be obscured by the heart (fig. 21).

*Consolidation* results either from inflammation or from growth. In acute inflammations, such as *pneumonia* or *pneumonitis*, there may be an opacity of the whole lobe, or the shadows may be irregular and diffuse, as in *bronchopneumonia*. These shadows are quite non-specific and they may be identical with those cast by tuberculous areas. In more chronic cases *tuberculosis* is the common cause of an inflammatory shadow and the consolidation is usually irregular and not very dense. The term "infiltration" is applied to these (fig. 39). When there is chronic tubercle there is often fibrosis and calcification as well. In most chronic cases it is usual to find more than one of these processes in the same film, i.e. consolidation, cavitation, fibrosis, and calcification, and it is the combination of appearances which enables a diagnosis of tuberculosis to be made. Other chronic infections, such as actinomycosis and syphilis, may give rise to similar appearances, except that calcification does not occur. It must be remembered that tubercle is a frequent complication of other chronic chest conditions.

Fibrosis is rarely so massive as to cause a dense shadow in the lung. The usual appearance is that of localized haziness with increased striation, and the *mediastinum* is displaced towards the affected side (fig. 14). When large bronchiectatic sacs are present, these may appear as translucent areas in contrast with the denser fibrotic markings. Diffuse bilateral fibrosis is seen in occupational diseases, such as silicosis, in which there are various stages, ending in dense nodule formation (figs. 45, 46).

*Calcification* is characteristic of *healed tuberculosis*. The patches are usually sharply defined and opaque, in contrast to the softer shadows of consolidation (fig. 44).

*Tumours* may be innocent or malignant. *Innocent tumours* are rare and they are always sharply defined. They may be seen in any part of the lung field; those which arise actually in the lung are usually *chondromata*, which may show calcification. Innocent tumours in the lung or mediastinum must be distinguished from neurofibromata arising from an intercostal nerve. It is essential to have a good lateral view in these cases, and it may be necessary to induce a *pneumothorax* in order to

determine whether the tumour is attached to the chest wall or whether it moves inwards with the lung.

*Malignant tumours* arising directly from the lung tissue are extremely rare, and the diagnosis can only be made with certainty by histological examination. The great majority of malignant tumours take their origin in the bronchi and are, in fact, *bronchial carcinomata*. Four types are found.

1. The tumour which completely occludes a bronchus and causes collapse of that part of the lung supplied by the affected tube (figs. 13, 22). The picture is that of massive collapse of a lobe or of a lung. In such cases it is obvious that the extent of the X-ray change is no guide to the actual size of the tumour.

2. The tumour may arise from a bronchiole and may be found in the substance of a lobe. In such cases the shadow is usually rounded, but the margins are indefinite (fig. 27). Should necrosis occur, a cavity with a fluid level may be seen, and the appearance may closely simulate a simple lung abscess.

3. A tumour arising from one of the bronchi, near the hilum, may infiltrate directly into the mediastinum, usually in the upper part of the chest, and it may therefore present the appearance of a primary mediastinal tumour or an aneurysm. Without further investigation it is impossible to be sure of the origin of the growth in such a case (fig. 29).

4. A tumour which arises in the bronchus may, and very often does, extend simultaneously in both directions, so that there is an irregular fan-shaped projection spreading out into the lung as well as the mediastinal mass. The mediastinal tumour may be continuous with the primary growth or it may consist of secondary deposits in the regional glands.

Secondary deposits may be either single or multiple, but much more commonly the latter. The nodules are round and sharply defined and they vary greatly in size (figs. 33, 34). Multiple shadows are characteristic of secondary growth, but a single tumour may be impossible to distinguish from a cyst or from a primary growth. Frequently the ribs can be seen to contain small deposits when a film is exposed with a Potter-Bucky diaphragm. In any case of doubt a search should be instituted for a primary growth elsewhere, the common sites being the breast, kidney, prostate, and thyroid gland.

Hydatid cysts may occur in the lungs. These vary greatly



in size and may be single or multiple. They are most commonly seen in the right lower lobe, and in many cases it is found that the liver is also affected. The cysts appear as round, sharply-defined areas which are homogeneous, and there is no fluid level. They have, however, no diagnostic characters; sometimes the walls are seen to be calcified.

5. *The pleura.*—Normally the lungs extend to the chest wall and the pleural membrane is not visible, but when disease is present there may be certain changes.

In *chronic pleurisy* of any sort the pleura may be considerably thickened, and in such cases there is a hazy appearance of the lung field. In tuberculosis the change is commonly at the apex, whereas when the thickening has followed a pleural effusion or an empyema the haziness is usually at the base. In such cases the normal sharp outlines of the diaphragm and the costophrenic sinus may be obliterated. Very rarely, *calcification* may occur in the pleura, and it is seen in the form of dense linear and spotted areas. It may result from the healing of tubercle, or an old-standing haemothorax or empyema.

*Pleural effusion* is not as a rule very difficult to recognize. When the effusion is small, there is an opacity which decreases in density from below upwards (fig. 47); it must be noted that the density of the opacity is not a reliable guide to the quantity of fluid present. When the effusion is larger, the opacity increases in size and the inner border tends to run downwards and inwards, but it is not always sharply defined. Usually the border is concave towards the mediastinum, but it is sometimes convex. When large effusions are present the heart and trachea are displaced away from the affected side (fig. 11). Should there be air in the pleural cavity, the upper border of the effusion *always* shows a fluid level which extends out to the thoracic wall (fig. 49). Even when air cannot be seen in the pleural cavity, a fluid level *always* indicates that air and fluid are present.

*Pneumothorax* is a condition which is very commonly seen in X-ray films, and it should be easily recognized in most cases. The deflated lung has a greater density than normal and the visceral pleura can be seen as a sharp line. Outside this is the pleural space, which is an absolutely clear area *not traversed by any lung markings* (fig. 12); when there is a large quantity of air present, the heart and mediastinum may be displaced

towards the opposite side. In spontaneous pneumothorax it is always essential to examine the lung fields very carefully in order to be sure whether tubercle or other disease is present.

*Tumours* of the pleura do not show very definite shadows in the film. There may be an appearance of gross thickening, or of fluid, but there is no local mass as a rule. Subpleural tumours are usually *neurofibromata* arising from an intercostal nerve and projecting as spherical, sharply defined masses into the lung field. It is usually impossible to be sure whether such a tumour is in or outside the lung, and an artificial pneumothorax may be necessary to make certain. A lung tumour will retract with the collapsing lung, whereas a subpleural tumour will be seen projecting from the chest wall into the clear pleural space.

### Lipiodol

A bronchogram is sometimes of the utmost value in reaching a diagnosis, and as the technique is comparatively simple, it will be described in detail.

Before lipiodol is given to any patient it is necessary to test for sensitiveness to two drugs, iodine and cocaine. In the first place, a subcutaneous injection of  $\frac{1}{15}$ th grain of cocaine is given. The pulse is watched at intervals for half an hour, and any rise in rate, or tendency to faintness, indicates that the patient is sensitive to the drug. Should cocaine be administered to a patient who is sensitive in the quantity used while giving lipiodol, alarming and occasionally fatal collapse may occur. Sensitiveness to iodine is tested by giving 30 grains of potassium iodide daily for two days. Should the patient be sensitive, there may be an acute catarrh of the nose and mild conjunctivitis, together with a skin eruption. It is unwise to pursue a lipiodol investigation in patients who are demonstrably sensitive to iodine, but the patient may be tested for sensitiveness to bromide, and, if this test is negative, a bromine-containing oil (Bromipin) may be substituted with fairly good results.

The oil must always be warmed to body temperature before injection. This renders the process less uncomfortable for the patient and also makes the oil less viscid, so that it is more easily introduced. The quantity injected varies with the condition which is being investigated. A normal lung, or one with mild bronchiectasis, is adequately filled by 10 or 12 c.c.

of oil. When there is gross bronchiectasis, from 20 to 40 c.cs. may be necessary. The amount may be judged by screening before the films are taken. When a lung is completely collapsed, not more than 5 c.cs. should be injected, otherwise collapse of the opposite lung may occur and severe dyspnoea may result. *Films should be always taken in the lateral as well as the antero-posterior position unless the oil has been injected into both sides at the one operation.*

There are three methods by which lipiodol may be introduced.

*The nasal route.*—For this examination it is necessary to have a de Vilbiss spray, a 20-c.cs. syringe fitted with an olive-shaped nozzle of such size that it fits securely into the end of a gum-elastic catheter, a 2-c.cs. syringe, and a few c.cs. of a 10 per cent solution of cocaine. The catheter is sterilized by means of formalin vapour and should be lubricated with glycerine before use.

The patient sits upright and the selected side of the nose is sprayed with the cocaine solution. Either side of the nose may be used, but it is advisable to be sure that a deflected septum or any other gross abnormality does not cause obstruction on the side through which the catheter has to pass. The throat is then anaesthetized in a similar manner. The de Vilbiss spray, which has an adjustable nozzle, is the most suitable for this purpose, for the posterior pharyngeal wall, the back of the tongue, and the larynx may each be dealt with in turn. Five minutes are allowed to elapse for full anaesthetization to take place, and a No. 5 gum-elastic catheter is then introduced into the nose. It is pushed on until it can be felt to impinge upon the posterior pharyngeal wall, and to turn downwards. The head of the patient is controlled by the operator and is now bent slightly forwards. The patient is instructed to breathe regularly and deeply, and the catheter is pushed gently onwards during inspiration, when it will be found to slip quite easily into the larynx. It may now be pushed down to its full extent and will come to rest in one of the main bronchi. It is astonishing that the catheter enters the larynx so regularly, and it is a fact that failure, in expert hands, is comparatively rare. There is usually a slight cough as the catheter passes through the larynx, and phonation is altered or lost. Should there be any doubt whether the catheter is really in the trachea, the laryngeal

mirror may be used to make certain. As soon as the catheter is in position, 1 c.c. of the 10 per cent cocaine solution is injected through it in order to numb the mucous membrane of the trachea.

The position of the patient during the injection is determined by the part which it is desired to fill. For the lower lobes the patient should be upright, whereas for the upper lobes recumbency is necessary. The great advantage of this method is that the oil can be injected while observations are made on the X-ray screen, thus ensuring proper filling of the required area and avoiding the risk of overfilling. It is sometimes desirable to take pictures both recumbent and erect, and when there is evidence of abnormality it is essential that lateral views should also be taken. For this purpose it is important to remember that one side only should be filled before the lateral view is taken, otherwise the bronchial markings on the two sides will be confused. The catheter is usually left *in situ* until the film has been taken, and it may then be removed. *The patient should be instructed not to swallow any food, either liquid or solid, for at least two hours after the cocaine has been given, as otherwise there is a risk of inhalation of foreign material into the bronchi.*

*The oral route.*—This is the simplest method, but it is not reliable when filling of the upper lobes is required. It is often possible to introduce the oil into the upper bronchi, but this cannot be guaranteed. In addition, it is not uncommon for some of the oil to pass down into the stomach, and it is therefore not until the patient is screened at the end of the operation that it is known whether a successful result has been obtained. For these reasons this method is definitely inferior to the nasal route, but its greater convenience makes it the method of choice in some cases.

The instruments required are a throat spray, about 5 c.c.s. of 10 per cent cocaine solution, some tongue cloths, and a 20-c.c.s. syringe to which is attached a cannula, which may be straight or slightly curved at the end, and about three inches in length. The patient sits upright, with the head bent slightly backwards, and the fauces, pharynx, and larynx are sprayed with cocaine. After five minutes the syringe is filled with lipiodol and the patient is instructed to breathe evenly and deeply, making a slight noise on inspiration. The lipiodol is poured slowly over

the base of the tongue and, in a patient who co-operates well, it will drop into the trachea. The patient should be inclined towards the side which it is desired to examine. Immediately after the oil has been introduced the patient may be instructed to lie on one side for a few minutes if it is desired that some should enter the upper bronchi. The patient is then screened and films taken.

*The crico-thyroid route.*—This method approaches more nearly to a surgical operation and is best avoided in nervous patients. There are also certain disadvantages to the method. In the first place, it is possible that infected material might gain entrance from the puncture in the crico-thyroid membrane into the tissues of the neck, setting up a severe, and perhaps fatal, cellulitis. Unless due care is taken, it is also possible to inject the lipiodol into the tissues of the neck and upper mediastinum, and this, although not necessarily harmful in itself, leaves permanent radiographic evidence of bad technique.

The instruments required are a 2-c.cs. syringe, a hypodermic needle, an intravenous needle, a special trocar and cannula (that described by Chandler is the most suitable), 2 per cent novocaine and 10 per cent cocaine solutions, and a 20-c.cs. syringe.

It is most convenient to carry out the operation with the patient recumbent. For the preliminary stages a small pillow is placed under the neck, thrusting the larynx and trachea forward. The region of the crico-thyroid membrane can be easily felt in the space immediately below the cartilaginous part of the larynx, in the mid-line. A small amount of novocaine is injected into the skin immediately over the membrane and then, with the intravenous needle, the membrane itself is anaesthetized and punctured. It is easy to tell when the membrane has been punctured because air can be drawn into the syringe without resistance. Leaving the needle *in situ*, the small syringe is then filled with  $\frac{1}{2}$  c.c. of 10 per cent cocaine solution, and this is injected directly into the trachea. The needle must be withdrawn immediately, as there is always a reflex cough and it is possible that the needle might be broken. (It is maintained by some that cocainization of the trachea is unnecessary and it may be omitted if desired.) After two or three minutes the trocar and cannula are pushed through the crico-thyroid membrane into the trachea and the trocar is removed. The

20-c.cs. syringe is then attached to the cannula and the piston is withdrawn in order to be sure that the end of the cannula is in the trachea. The patient is made to lie on the affected side and the lipiodol is injected slowly, the piston of the syringe being withdrawn slightly after every two or three c.cs. have been injected in order to make sure that the end of the cannula is still in the trachea. If this precaution is observed, lipiodol cannot be injected into the tissues of the neck. When the requisite amount of oil has been instilled the cannula is withdrawn and the puncture sealed with collodion.

This technique is the most certain of the three and in skilled hands it is the method of choice.

Toxic symptoms do not appear to arise as a result of absorption of the oil from the bronchi, and most of it is coughed up in the course of a day or two. If screening shows that any appreciable amount has been swallowed, it may be desirable to wash out the stomach as a precaution, although this is by no means always done and most patients come to no harm. A dose of salts is probably all that is really necessary.

Sometimes lipiodol enters the alveoli, where it may remain for months, or even years. No harm results so long as the fact is recognized in subsequent films. Residual lipiodol has sometimes been erroneously reported as tuberculous infiltration, but it is always diffusely scattered in the alveoli, and the individual outlines are quite sharply defined; the whole shadow has a hard and metallic quality in contrast to the softness of tuberculous infiltration. Close inspection of the film, and knowledge of the past history, should enable the true interpretation to be reached (fig. 40).

The interpretation of lipiodol films is quite easy so long as the normal appearances can be recognized (figs. 7, 8). The significance of abnormalities, and their appearance, is considered in the section which deals with the various diseases.

Lipiodol examinations should be carried out in the following circumstances:—

- (a) To determine the presence of bronchiectasis and to define its extent.
- (b) To make sure that there is no bronchiectasis in the apparently sound lobes. This is essential when lobectomy is being considered.
- (c) When it is necessary to be sure whether a cavity is in

communication with a bronchus. A bronchiectatic cavity should fill with lipiodol, whereas a lung abscess usually does not. It is of no value in the distinction between simple lung abscess and breaking-down growth.

- (d) To determine the presence of bronchial obstruction and to locate it. This is especially important in suspected early carcinoma.
- (e) It is sometimes useful to inject lipiodol through a sinus in the chest wall ("pleurogram"). This is done in cases of chronic empyema in order to define the size and position of the cavity, and it is most useful in showing the presence of a broncho-pleural fistula. If lipiodol is injected into the pleural cavity and the film shows the presence of oil in the bronchi, then a *broncho-pleural fistula* is certain to be present (fig. 51).

### Diagnostic Pneumothorax

It is sometimes uncertain, from examination of plain films, whether a lesion is situated in the lung or outside it. In such cases the routine is to induce an artificial pneumothorax and then to take further films for comparison. If it be found that the shadow moves inwards with the collapsing lung, it may be assumed that the lesion is in the lung itself. When, however, there is a tumour in a rib, or attached to the parietal pleura, the shadow remains stationary and the lung is seen to move away from it. Tumours in connection with the visceral pleura can sometimes be seen to stand out clearly, projecting into the air space.

### Tomography

This term is used to indicate the investigation of a selected layer of the chest. The ordinary plain film is a composite picture of the whole thickness of the chest and it is therefore possible that lesions of considerable size will fail to be recorded should they be situated near the front or back of the chest; a cavity in the centre of a solid area is very likely to be missed.

In tomography there is a deliberate diffusion of all superfluous structures, so that the condition in a given plane can be visualized. This diffusion is brought about by a movement of the X-ray tube, grid, and film about the plane to be shown during the exposure. The result is a general vagueness about the appearance of the film, with any lesion in that particular plane standing out clearly (figs. 9, 10). In chest work it is

customary to make exposures centred in six or more planes from behind forwards and thus unsuspected disease may not only be brought to light but also accurately localized.

### Kymography

The kymograph is an X-ray instrument in which there is a metal grid with a row of transverse slits of equal width and equidistant from each other. This is placed between the patient and the film cassette and, during the exposure, it is moved at right angles to the slits. The result is a picture in which the different phases of the cardiac movement are separated instead of being superimposed on each other, as occurs in the plain film. Any moving border appears as a serrated edge and it is possible to distinguish between expansile and transmitted pulsation. So far as respiratory diseases are concerned, the kymograph is of use in cases where there is a doubt whether a mediastinal shadow is due to a tumour or an aneurysm.

### "Double-Exposure" Films

The great advantage of screening is that the respiratory movements can be watched. Quite recently a method has been developed by which a record of this excursion can be preserved. Two exposures are made on the same film on full expiration and inspiration. The radiogram can be studied, the respiratory excursion can be measured, and a record can be preserved. Diminished movement may be demonstrated in emphysema and in asthma. This type of film is useful in assessing effect of breathing exercises.

### Miniature Mass Radiography

The apparatus which is used in miniature mass radiography consists of an X-ray screen with a camera attachment by means of which the image on the screen is photographed. The camera contains a roll of film on which exposures can be made at frequent intervals, so that a large number of patients can be examined in a short time. When the film is developed it is placed in a projector and a magnified image of the chest is



thrown on a screen. Even quite trifling abnormalities are detectable by this means. Whenever the appearance is considered to be abnormal the patient is recalled for radiographic examination on a full-sized X-ray film.

Large scale examinations are being carried out on various sections of the population, the Services, factory workers, and the Civil Service. The results are as yet far from complete, but it is possible to state that miniature mass radiography is a most useful means of detecting early respiratory disease. There is a widespread misconception that the method is only of value in searching out cases of tuberculosis, but this is, of course, quite erroneous. Any variety of chest disease which would be evident on a full-sized film will be revealed by miniature radiography, and emphysema, fibrosis, bronchiectasis, tumours, and various cardiac conditions are commonly found.

The practical utility of the method is beyond doubt and it is likely to be used increasingly as further units become available. The great majority of the patients who are found to have some early lesion have no symptoms, and experience shows that most of them have no recognizable physical signs. This is, therefore, the best and most reliable means of detecting early respiratory disease available at the present time,

From the clinical point of view there are two difficulties which have been found to arise in practice. Many patients who have a radiological abnormality have no symptoms, and it is not always easy to separate those for whom treatment is essential from those who may be safely allowed to carry on at work. This applies especially to cases of *tuberculous disease*, in which there is often difficulty in deciding whether the lesion is active. It is frequently necessary to keep a patient in this group under regular X-ray observation before a decision can be reached.

The large number of cases of early tuberculosis which have already been discovered has thrown a considerable strain on sanatorium accommodation. This is a serious difficulty, but it is clear that the search for early cases of tuberculosis must be carried on.



of these same conditions, and the amount may reach ten or even twenty ounces daily. Very occasionally it happens that a large quantity of pus is coughed up suddenly. This may occur when a lung abscess, an empyema, or a subphrenic abscess, ruptures into a bronchus.

The expectoration of a large quantity of watery, frothy sputum is characteristic of *acute oedema of the lungs*, and of the rare condition, *bronchorrhoea serosa*. The sudden appearance of a large quantity of comparatively clear liquid is very suggestive of the rupture of a hydatid cyst; the presence of fragments of daughter cysts is of material help in establishing the diagnosis.

(b) *Appearance*.—The appearance of the sputum is of considerable diagnostic importance. In the first place, it is necessary to be sure that the material inspected is in reality sputum, and not saliva or secretion from the naso-pharynx. As a rule the patient can judge the origin of the material, but this is not always the case, and it may be necessary to note the manner in which it is produced.

*Watery sputum* is the result of *pulmonary congestion*, *acute oedema*, *bronchorrhoea serosa*, or ruptured hydatid cyst; it is never the result of an infection.

*Mucoid sputum* is clear in appearance and viscid in consistency. It is produced in the *larynx*, *trachea*, or *main bronchi* as a rule, and it indicates irritation of the respiratory tract, the commonest causes being *catarrh*, *excessive smoking*, the early stages of *acute bronchitis*, *chronic bronchitis*, *spasmodic asthma*, and conditions which result from the inhalation of dust. In *early tuberculosis* the sputum is usually mucoid, even although it contains tubercle bacilli, and specimens should therefore always be submitted to proper examination. A similar type of sputum is sometimes present in *bronchial carcinoma*.

*Purulent sputum* is yellow or greenish in colour. The degree of admixture with mucus varies greatly, and the intermediate stages between *mucoid sputum* and *pus* are termed "mucopurulent". Pus will be present when there is any well-established infection in the respiratory tract, and a bacteriological examination is necessary in order to identify the organisms concerned.

Other inferences may be drawn from the colour of the sputum. Grey sputum is not uncommonly present in city

dwellers and in smokers. A black specimen suggests the inhalation of carbon in large quantities, and is encountered in coal miners ("black spit"). The appearance of small yellow ("sulphur") granules in a purulent specimen is very suggestive of *actinomyco*sis, but the granules may easily be overlooked unless the specimen is carefully examined. A reddish colour, varying from brown to pink, indicates the presence of blood, which may be either fresh or altered in appearance according to the length of time which has elapsed between the actual bleeding and the production of the specimen. Fresh blood is easily recognized. The "rusty sputum" of lobar pneumonia consists of altered blood intimately mixed with tenacious mucus. A little blood mixed with pus may give rise to a pink appearance which is sometimes found in *bronchiectasis*. "Red-currant jelly" and "prune-juice" sputum are said to be found with bronchial carcinoma, but they are so rarely seen as to be of little value in diagnosis. Dark brown purulent material, alleged to resemble anchovy sauce, is seen in cases of amoebic lung abscess.

Certain other abnormal elements may also be noted. Discrete discs in a purulent specimen ("nummular" sputum) are said to indicate cavitation. Dittrich's plugs are small caseous masses, from the size of a pin-head upwards, greyish yellow in colour, and emitting an unpleasant odour when crushed, which occur in *bronchiectasis*. Curschmann's spirals in asthmatic sputum are more readily recognized under the microscope than with the naked eye. Finally, fibrinous casts of the smaller bronchi may be seen; they occur in *fibrinous bronchitis* and, rarely, in diphtheria. Fresh casts of the bronchi may be coughed up after a profuse haemoptysis.

It is said that a sample of sputum which separates into three layers on standing is characteristic of *bronchiectasis*, but the observation is of little practical value.

The odour of the sputum should always be noted. A foul odour always indicates infection with anaerobes, fusiform bacilli, or spirilla, and the conditions in which this occurs are certain types of lung abscess, *bronchiectasis*, and bronchial carcinoma.

(c) *Laboratory examination*.—When the sputum is copious any sample may be taken but, when it is scanty, and especially when it is essential to know the exact bacterial content, the

material produced in the early morning is most likely to yield satisfactory results. The patient must be instructed to wash out the mouth with a simple alkaline solution in order that the specimen may be obtained, so far as possible, free from contamination. It is desirable first to examine a specimen spread out on a glass slide. Sometimes it is possible to recognize fragments of lung tissue, or the "sulphur" granules of actinomycosis.

The fresh film must then be inspected under the microscope, and pus cells and red cells will be easily identified. In specimens from cases of spasmodic asthma Charcot-Leyden crystals are sometimes found. These are colourless, needle-shaped crystals which appear to be octohedral. Curschmann's spirals occur quite commonly in the sputum brought up during an attack of asthma and very rarely in any other condition. They are white or yellow in colour and are twisted into coils which may reach 1 cm. in length, rarely longer, but they cannot be recognized with certainty by the naked eye. Under the low power they appear as threads which have a clear central core around which fine mucinous fibrils are wound. Their exact composition is not known.

Elastic fibres may be seen under the low power, although high power examination is always desirable in order to establish their identity. They are more clearly demonstrated when the specimen is mounted in a drop of 10 per cent caustic soda solution. They are then seen as slender, refractile threads with a double outline, and frequently with curled or split ends. Sometimes they are seen in alveolar formation, retaining the original outline of the lung parenchyma, a point which proves their origin from lung tissue. The presence of elastic fibres in the sputum is proof positive of a lung lesion; they are most commonly found in *tuberculosis* and less often in *lung abscess* or in *malignant disease*. They should not be present in true bronchiectasis.

Evidence of parasitic infection is sometimes noted on routine examination. The only one likely to be found in this country is hydatid cyst, which is rare; the appearance of the typical hooklets is diagnostic of this disease. Very rarely the *entamoeba histolytica* or its cysts may be found, usually in cases where a liver abscess has ruptured into the lung. Naturally this condition nearly always occurs in patients who have been for

some time resident in the East. In Japan the ova of the lung fluke are commonly found, especially in cases of epidemic haemoptysis.

Appropriate staining of the films by means of methylene blue may show more detail of the structure of the cells in the sputum. This will give a general idea of the cells and bacteria present, and special stains may then be employed as indicated. Those in most common use are :—

For cells, Leishman's stain.

For bacteria, Gram's stain.

For tubercle bacilli, Ziehl-Neelsen's stain.

Examination of the film prepared with Leishman's stain shows the types of white cells present. Pus cells occur in every sputum which results from infection as opposed to irritation, and they are of no diagnostic significance apart from this. Eosinophil cells are found in considerable numbers towards the end of an attack of asthma and, in smaller numbers, in the intervals. Their particular significance is that they occur in cases in which the allergic element is important.

Epithelial cells are nearly always present and may come from any part of the respiratory tract. It is desirable when possible to recognize their probable site of origin.

Large flat polygonal cells with a comparatively small nucleus are squamous in origin and come from the upper air passages. They are frequently studded with bacteria and they have no pathological significance.

Ciliated cylindrical cells originate from those parts of the tract which are lined by a ciliated membrane; they usually come from the trachea and bronchi, although it must be remembered that similar cells are also present in the nasal mucosa. They are not very often identified, as they are usually much altered from their normal form when found in the sputum.

Occasionally large round or oval cells, with one or more nuclei, are encountered; these are thought to come from the alveoli of the lungs themselves. They nearly always contain particles of carbon, especially in city dwellers. In chronic heart disease, and in other conditions in which there is long-standing passive congestion of the lungs, they may also contain numerous brown granules of altered blood pigment, and they are then known as "heart-failure cells". Similar cells are often present for some days after a haemoptysis.

It is difficult to recognize malignant cells in the sputum with certainty. Large cells which stain deeply with methylene blue, which have dark, irregular nuclei, and which tend to be clumped, are suspicious of neoplasm, but absolute identification is still a matter for the expert.

*Bacteriology.*—The technique of the bacteriological examination need not be considered in detail. The routine examination consists of a Gram film, Ziehl-Neelsen film, and cultures on the appropriate media. In all cases of acute inflammation in the respiratory tract, and also in many cases in which there is gross infection, although the process is not acute, it has now become customary to incubate special cultures in order to test the sensitiveness of the bacteria present to penicillin.

### Penicillin

The introduction of penicillin is likely to result in a change of attitude in our approach to the problem of acute respiratory infections. In the past, the emphasis in teaching has always been laid on accuracy in diagnosis, and it is right that this should be so. We are now provided with a substance of proved efficacy in the treatment of many of the acute infections, and it is only natural to suppose that this will be used more or less empirically and that, in fact, it may come to be employed as a kind of clinical touchstone by means of which a diagnosis will be reached after observing the result of treatment. In cases of emergency this is quite justifiable, and it would be wrong to withhold penicillin from a desperately ill patient until the precise nature of the infecting organism is established. Nevertheless the virtues of penicillin must not be allowed to become an excuse for omission to carry out the proper investigation of the patient.

Penicillin is still a very recent addition to our therapeutic resources, and it is yet too early to be dogmatic about its action or about the results of treatment. At first it seemed that bacteria could be divided into two groups, those which are "sensitive" and those which are "insensitive". Experience has shown, however, that this is not quite true. In the group of apparently insensitive organisms it has been found that some are affected after higher doses than usual are given, and individual variation in sensitiveness is described. It is

therefore necessary in any case of doubt to test the sensitive-ness of the organism to penicillin before deciding to embark on treatment. This test is now carried out as a routine in any pathological laboratory, but a result cannot be expected in less than twenty-four hours.

*Penicillin sensitive organisms.*—It is fortunate that most of the organisms which cause the dangerous types of acute respiratory infection are sensitive to penicillin. The pneumococcus, staphylococcus aureus, streptococcus pyogenes, and most strains of streptococcus viridans are all sensitive, and therefore penicillin treatment is likely to be effective in cases of infection due to any of these organisms.

In addition to this, it is found that many anaerobes and spirilla, such as are commonly present in cases of lung abscess, are sensitive.

Another sensitive organism which is commonly present in respiratory infections, although it does not usually occur alone, is *Micrococcus catarrhalis*.

Among the less common infections due to penicillin sensitive organisms are *actinomycosis* and *anthrax*.

*Penicillin resistant organisms*—It is in this group that increase in the size of dose may alter our views as further experience is obtained. In the first place it must be stated that the *tubercle bacillus* is not affected by penicillin. The group of Gram-negative bacilli is relatively insensitive, and therefore penicillin is unlikely to be of value when the infection is due to *B. Pfeiffer*, *B. Friedlander*, or *B. pertussis*.

The majority of the virus diseases do not respond to penicillin, but it has recently been stated that some of the larger types of virus may be sensitive. *Psittacosis* falls into this group, but there is not yet any clinical evidence of the effect of penicillin in this disease.

*Penicillin administration.*—The route and dosage are not yet finally settled but the following is a brief summary of the methods in use at the present time

*Intramuscular injection.*—Continuous administration by means of a drip is now rarely employed. Perhaps the most usual method is repeated injections at intervals of 3 hours. For this purpose, from 20,000 to 30,000 units of penicillin are dissolved in from 2 to 4 c.c.s of isotonic saline solution. This method is reliable, but it suffers from the disadvantage that



eight injections are needed in twenty-four hours and thus there may be interference with the patient's rest. Sometimes the injections are painful and they may not be well tolerated after 3 or 4 days.

The most recent method is to employ a solution of 125,000 units in an oily suspension. The official mixture is :—

R

|                    |   |   |                  |
|--------------------|---|---|------------------|
| Calcium penicillin | . | . | 12,500,000 units |
| White beeswax      | . | . | 4.5 grammes      |
| Arachis oil to     | . | . | 100 c.cs.        |

The preparation should be warmed to blood heat for several minutes and shaken before drawing it into a warm syringe.

It is believed that an effective blood concentration is obtained which lasts for about ten hours with this dose. This method is not quite so easy to use from the point of view of the technique of injections, but it is undoubtedly less uncomfortable for the patient.

*Dosage.*—It will be seen that, by either of the injection methods, the average daily dose should be between 200,000 and 250,000 units. In acute infections treatment should be carried on for 4 or 5 days, so that an average total dose of about 1 million units is the rule. In severe cases, or when resistance is lowered, the course of treatment must be carried on so long as it is considered necessary.

*Inhalations.*—When the main infection is in the bronchi rather than in the lungs it appears preferable to administer penicillin in the form of a very fine vapour, such as can be readily obtained by several atomizers now on the market. In this way it is possible to treat cases of bronchitis and bronchiectasis with rather more effect than can be obtained by intramuscular injection. It is even possible to obtain an effective blood concentration from penicillin inhalation.

In cases of empyema 240,000 units of penicillin may be injected into the cavity and left *in situ* (p. 285).

*Indications for penicillin administration.*—Acute lung infection which is due to a penicillin sensitive organism may be treated efficiently by intramuscular injection. Thus, pneumococcal pneumonia and staphylococcal and streptococcal bronchopneumonia often respond in a dramatic manner. Of course, pneumococcal pneumonia usually responds quite well

to the sulphonamides, and there is not much to choose between the two methods of treatment in the average case. Penicillin, however, should always be used when there is no evidence of response to sulphonamide within twenty-four hours, or when there is any clinical evidence of sulphonamide poisoning or sensitivity; it is also much safer to use penicillin when there are complications such as heart failure, chronic renal disease, or leucopenia. Penicillin is undoubtedly more effective than sulphonamide in the treatment of streptococcal and staphylococcal infections.

In cases where there is a mixed infection, some of the organisms being penicillin sensitive, it would seem reasonable to employ this method of treatment, perhaps in combination with one of the sulphonamide group of drugs.

In acute and chronic bronchitis, in bronchiectasis, and in some cases of chronic lung abscess with copious sputum, it is reasonable to try the effect of penicillin inhalations. It must be understood that no final conclusions have yet been reached about the optimum dosage, and it may be that the next few years will bring considerable modifications in the present scheme of penicillin administration.

When dosage is being considered it appears to be quite safe to err on the side of giving too much, and it is certainly a great mistake to give too little, for by this means the organisms may become temporarily penicillin resistant. There have been some reports of ill-effect resulting from penicillin administration. Occasional allergic reactions, such as urticaria, arthralgia, and serum sickness and asthma, are on record, but these are hardly ever sufficient to contra-indicate the use of penicillin. It is probable that these ill-effects are due to the presence of impurities, and it is likely that they will no longer occur when a really pure product is used.

The organisms most commonly found in the sputum may be considered according to their staining properties.

#### 1. Gram-positive Cocci

(i) *Pneumococcus*—This is the commonest of all the organisms found in the respiratory tract and it is present in

large numbers in the rusty sputum of *lobar pneumonia*. It is often met with in cases of *bronchopneumonia*, *bronchitis*, in some cases of *lung abscess*, and as part of a mixed infection. The organism is also found in the mouth and nose in healthy people and its presence does not necessarily imply disease.

The pneumococcus is typically lanceolate in shape, lying in pairs with the blunt ends in apposition. Each pair is surrounded by a gelatinous capsule which can be seen as a clear halo in the Gram film, and which can be demonstrated by special methods. Cultures may be made on blood agar, when small, round, translucent colonies are formed.

Serological agglutinations have shown that pneumococci can be separated into no less than 32 distinct types. The common organisms to be found in *lobar pneumonia* are of types 1 and 2; type 3 is a more virulent, but less common, organism which resembles a streptococcus in many ways and is sometimes termed "*streptococcus mucosus*". The remaining pneumococci are classed together as sub-groups of type 4, which is a composite group of relatively low virulence.

(ii) *Streptococcus*.—Any of the varieties of streptococcus may be found in the sputum, and the appearance of a chain of Gram-positive cocci in the film should indicate the necessity for a culture, which must be made on a blood medium, in order to identify the haemolytic types of streptococcus. These organisms cause acute disease such as *bronchopneumonia*, *lung abscess*, *empyema*, and the lung conditions which are found as a part of a general septicaemia.

Non-haemolytic streptococci are present in the sputum in many cases of subacute and chronic infection anywhere in the respiratory tract. They are not usually very virulent.

(iii) *Staphylococcus*.—This organism may be present as a contamination, and it is better not to accept it as being responsible for an infection unless repeated cultures have shown its presence. It may be the cause of *lung abscess*, especially when this is secondary to pyaemia. At other times it occurs simply as a secondary invader.

## 2. Gram-negative Cocci

Several kinds of Gram-negative cocci are found in the upper respiratory tract, but the majority are not of pathological significance. It is true that the meningococcus has its habitat

in the naso-pharynx, but this organism is seldom, if ever, the cause of local disease and it need not be further considered here. The only organism in this group which is definitely associated with respiratory infection is *Micrococcus catarrhalis*. This organism is a large Gram-negative coccus, frequently found in clusters, and intracellular. It can easily be identified by culture, for it yields distinctive, sticky, greyish-yellow colonies. It is present, not as a rule alone, in many acute and chronic bronchial infections.

### 3. Gram-negative Bacilli

(i) *Friedlander's bacillus*.—This organism is of interest in that it was the first to be described as the cause of pneumonia, but it is only found in a small percentage of cases. It is sometimes seen, in association with other organisms, in subacute or chronic infections. The organisms are rather long, non-motile, encapsulated rods which grow well on ordinary media and form a characteristic "candle-grease" colony.

(ii) *H. influenzae* (Pfeiffer's bacillus).—This is a minute Gram-negative rod which was presented as the cause of influenza. It is certainly frequently found in the respiratory secretions in influenza, but it is not the cause of the disease. It is found in many cases of chronic respiratory infection, and it is sometimes the cause of sinusitis with recurrent descending infection of the respiratory tract. Cultures must be made on a blood medium, when minute "dew-drop" colonies are formed.

(iii) *H. pertussis* (Bordet's bacillus).—This organism closely resembles Pfeiffer's bacillus, from which it can only be distinguished by its agglutination reactions. It is the cause of whooping-cough.

### 4. Gram-positive Bacilli

These are the least common of the organisms found in the respiratory tract.

(i) *C. diphtheriae* (Klebs-Loeffler bacillus).—This is present in the throat and nose in cases of true diphtheria and in carriers. Very rarely a descending infection may occur and the trachea and main bronchi may be covered with false membrane. The organism is usually identified in cultures made on blood serum, either of material taken from the throat by a swab or from portions of expectorated false membrane. Should the clinical

condition be doubtful it is necessary to confirm the virulence of the organism by means of guinea-pig inoculation.

(ii) *Anaerobic bacilli*.—These organisms usually appear in the sputum as Gram-positive rods of varying size. Their true nature can be suspected from the Gram film, but positive confirmation should be sought from anaerobic cultures. They occur in some cases of *lung abscess* and *bronchiectasis*, and sometimes in an empyema.

### 5. Other Organisms

(i) *B. tuberculosis*.—This is a medium-sized bacillus which can only be stained with difficulty. It is in fact Gram-positive, but the Ziehl-Neelsen stain is used as a routine for its detection. A suitable portion of sputum, preferably from a part which is purulent, is smeared thickly on a slide and is then dried and fixed by heat. The slide is immersed in a bath of steaming strong carbol fuchsin solution for five minutes. It is then decolourized, firstly in 25 per cent sulphuric acid for one minute, and then in 70 per cent alcohol until the stain ceases to come out, after which it is washed in water and counter-stained with methylene blue. The waxy envelope of the tubercle bacillus retains the pink dye and the organism can be readily picked out against the blue background. The reason for the use of acid and alcohol as decolourizing agents is that by this means the tubercle bacillus can be distinguished from certain similar bacteria. The bacillus of leprosy does not retain the stain when exposed to the acid, and the smegma bacillus is decolourized by alcohol. Certain non-pathogenic bacilli, such as the Timothy-grass bacillus and the butter bacillus, are also acid-fast; these latter organisms might be found in the stomach washings.

The tubercle bacillus is often slightly curved at one end and it may show beading or clubbing; it is allied, not to the lower orders of bacteria, but to the streptothrix group (*Mycobacterium tuberculosis*).

Tubercle bacilli may live for a long time in dry surroundings but they are rapidly killed by exposure to sunlight and by strong antiseptics. In the dry state they are comparatively resistant to heat, but when moist they are killed at a temperature of 60° C.

Culture is difficult on ordinary media, but the organism

may be grown on Dorset's egg medium or in glycerine broth ; growth appears after about ten days' incubation at 37° C. A better medium is that devised by Lowenstein ; it contains asparagine and is enriched by the addition of Malachite Green, and has proved satisfactory for the culture of sputum, pleural fluid, and other material. The final test of the nature of a doubtful organism is guinea-pig inoculation, for the tubercle bacillus alone is capable of producing the lesions of tuberculosis.

The two important types of tubercle bacillus are the *human* and *bovine* forms, which are culturally distinct and which behave differently in the body. The human type grows fairly easily (eugonic), producing a dry, yellowish, rough growth, whereas the bovine grows with difficulty (dysgonic), and forms a white, moist, smooth surface. When inoculated into rabbits the human bacillus produces a local lesion and the bovine causes fatal generalized tuberculosis.

The role bacillus has been extensively studied in this country and it has now been accepted as a variety of tubercle bacillus. Its great importance lies in the fact that it appears to have a considerable power of producing immunity against infection with the human tubercle bacillus, and it may turn out to have several advantages over B.C.G. vaccine in this respect (p. 211).

(ii) *Streptothrix*.—The ray-fungus, *Streptothrix actinomyces*, is an organism which consists of a mycelium, the threads having club-shaped extremities. There may be a macroscopic granule which, under the microscope, is seen to consist of a central dense felt-work of filaments which show true branching but no spores. Radiating from the centre are the typical clubs which are thought to represent either a degeneration or a defensive reaction, more probably the latter. The filaments stain Gram-positive, but the clubs are Gram-negative as a rule. The organism grows in anaerobic culture in glucose blood broth. It may be found in sputum or in pus from an empyema.

(iii) *Yeasts and fungi*.—Many organisms are included in this group, but few are found in the respiratory tract. *Aspergillus* is occasionally seen in the sputum, but usually it is only a saprophyte in a tuberculous or bronchiectatic cavity ; cases of primary infection of the lung tissue are very rare. Similar observations apply to yeast-like organisms (monilia) and other fungi, such as the sporothrix, which have been reported in the

sputum, but which have rarely been shown to have caused lung disease.

(iv) *Fusiform bacilli and spirilla*.—It has long been known that ulceration of the throat and gums may be due to a combination of fusiform bacilli and spirilla. It is now recognized that the foul odour of the sputum in certain cases of *lung abscess*, *bronchiectasis*, and *growth* results from infection with organisms which appear to be identical. The combination is characteristic, and can be readily demonstrated in a Gram film of the sputum, but the best results are obtained when Leishman's stain is used. The bacilli are thin Gram-positive rods which vary greatly in length and may be beaded; the spirilla are delicate Gram-negative threads with wide, irregular curves. Culture is difficult and is not necessary in clinical work.

(v) *Rare organisms*.—Infection of the respiratory tract is nearly always due to the bacteria already described, but occasionally some other organism may be found. Atypical inflammations are sometimes due to bacilli of the typhoid group, or to *B. melitensis*, and a severe bronchopneumonia may result from infection with *B. pestis*, the cause of plague, or from *anthrax*.

The part played by *filterable virus* in the causation of chest disease is not yet clear. There seems no reason to doubt that the *common cold* and *influenza* are due to this type of infection. Recently it has been suggested that this agent is also responsible for "atypical pneumonia", but its relation to this common disease is not yet fully determined. A few years ago there was a small epidemic of *psittacosis*, with severe and often fatal bronchopneumonia, and the pathological evidence pointed convincingly to a virus as the cause of the disease.





*respiratory condition is syphilitic in origin.* In many cases of bronchial carcinoma, for instance, the test is positive, and the clinical distinction from gumma of the lung may be extremely difficult. In suspected *aneurysm* a positive reaction is of some confirmatory value, but the clinical and radiological evidence must always be considered together with the result of this test. In cases where it is impossible to resolve a doubt about a diagnosis between syphilis and malignant disease, it may be justifiable to observe the effect of an intensive course of anti-syphilitic treatment before forming a final opinion.

(c) *The erythrocyte sedimentation rate.*—This is a very simple test to perform. Several different methods are described but that of Westergren is most commonly employed. A standard glass tube, graduated up to 200 mm., and of 2.5 mm. internal bore, is used for the test. A 3.8 per cent solution of sodium citrate is used to prevent clotting and 0.4 c.c. of this is drawn into a syringe. Blood is then withdrawn from a vein up to the 2.0 c.cs. mark and, after being thoroughly mixed, is expelled into a small glass tube. The citrated blood is drawn up to the 200 mm. mark in the Westergren tube which is then fixed upright in a rack. The changes which occur at the end of one and two hours are recorded in terms of millimetres of clear plasma which are visible above the column of sinking red cells. Normally the sedimentation rate is 5 mm. or less in the first hour.

The sedimentation rate is increased in any condition which is associated with *toxaemia*, and the rate of fall is quite a good indication of the degree of toxaemia present. *But the test is of no value at all in the diagnosis of any particular infection.* In active tuberculosis, for instance, the rate may be normal or greatly increased and the test is used to indicate the need for rest. A series of observations is of great value in following the progress of any infection; it is never wise to allow a convalescent patient to get up while the sedimentation rate is much above normal. In tuberculosis the response to treatment, and consequently the prognosis, can be assessed with considerable accuracy in this way. An unexpected increase in the E.S.R. is often a warning that some complication is about to declare itself.

## Pleural Fluid

In every case of pleural effusion a small quantity of the fluid, about 20 c.cs., should be removed for examination. When the fluid is clear, half should be mixed with a little sterile 2 per cent citrate solution in order to prevent clotting, and so to assist the cytological examination.

Fluid in the pleural cavity may be clear, purulent, blood-stained, or chylous.

Clear effusions are divided into *exudates* and *transudates*. The differences between the two types may be tabulated as follows :—

|                                | Transudate               | Exudate  |
|--------------------------------|--------------------------|--|
| Appearance . . . . .           | Straw-coloured           | Straw-coloured   |
| Spontaneous clotting . . . . . | Does not occur           | Usually  |
| Specific gravity . . . . .     | Less than 1014           | 1014 or over   |
| Protein . . . . .              | Less than 1 per cent     | More than 1 per cent                                     |
| Cells . . . . .                | Few, chiefly endothelial | Usually many lymphocytes; polymorphs are sometimes found |

From a consideration of these points it is usually possible to decide whether a given fluid is an exudate or a transudate. The causes of the various types of pleural effusion are considered later (p. 274).

The cell content of the fluid is next studied. Red cells often result from trauma at the time of puncture, but they occur also in *tubercle* and *growth*. The presence of *polymorphonuclear* cells indicates *pyogenic infection*, but *organisms* need not necessarily be present, for a cellular exudate may occur in the pleura as a result of inflammation in the surrounding organs, even under the diaphragm. *Lymphocytes* suggest the presence of *tubercle*, although they may occur with other chronic inflammations. *Endothelial* cells are shed into the fluid from the pleural membrane and a few may be found in transudates. Their presence in quantity suggests malignant growth. Sometimes these cells are altered in appearance, larger and more deeply-staining, with more than one nucleus; such cells are termed *Fouli's cells* and, although not themselves of malignant origin, they are said to be found when a growth involves the pleura.

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The bacteriological investigation consists in the examination of a Gram film for the common organisms and confirmation by culture. A Ziehl-Neelsen film may reveal the presence of tubercle bacilli, but these are more likely to be found by culture of the fluid or by guinea-pig inoculation. When pus is found it is submitted to the ordinary methods of examination by films, cultures, and animal inoculation where necessary. In acute cases pneumococci, streptococci, staphylococci, or anaerobes are most commonly found. Tubercle is a common cause of chronic empyema but, if it proves impossible to find the bacilli, search for some form of streptothrix should be instituted.

Fluids which are deeply blood-stained seldom yield any information of value on pathological examination.

Opalescent fluids are rarely met with. They may be *chylous*, or *chyliform*. Chylous fluid contains true chyle, and always results from damage to the lymphatic system; under the microscope fatty globules will be seen. Chyliform fluid is homogeneous under the microscope, for it contains lipoid; it occurs in some cases of hydraemic nephritis and in effusions of long standing. Chemical analysis is the most certain method of identifying the type of fluid.

## CHAPTER 12

### SPECIAL DIAGNOSTIC METHODS

#### Bronchoscopy

WHEN the diagnosis has not been made with certainty as a result of the foregoing investigations, it may be necessary to apply certain further methods, all of which require special skill and will not therefore be discussed in detail.

The bronchoscope is employed on an extensive scale in diagnosis and, to a less extent, in treatment. It is now part of the routine investigation of the obscure chest case. The great value of this instrument is that it enables the mucous membrane of the main bronchi and part of their primary divisions to be inspected and material removed for examination, both histological and bacteriological.

Bronchoscopy should only be carried out by an expert, for the technique is not easy and the interpretation of the appearances requires experience. A preliminary injection of morphia and scopolamine is given and the throat and larynx are well anaesthetized with cocaine. It is surprising how well most patients tolerate the passage of the instrument. A general anaesthetic is rarely necessary.

The main indication for bronchoscopy is in diagnosis. At first it was employed chiefly for the location and removal of *foreign body* but it is now regarded as essential in the investigation of any suspected disease in the larger bronchi. The diagnosis of *bronchial carcinoma* is best confirmed by this means—the growth can often be seen and a piece removed for microscopic section. Bronchoscopy is the only sure means by which an early carcinoma can be recognized. Increasing experience has shown that about 10 per cent of bronchial tumours are not malignant, and it is therefore of the utmost importance that the exact nature of the growth should be established by this means before the plan of treatment is finally decided. Inflammatory lesions can also be studied at first hand. It is established that tuberculous ulceration is fairly common in the larger bronchi, while other granulomatous conditions are sometimes seen. Occasionally a stricture,

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either tuberculous or syphilitic, may be encountered and bronchoscopy is essential in the distinction between these lesions and carcinoma. An oedematous appearance of the mouth of a bronchus and the observation of pus exuding from it are valuable indications of the exact position of a *lung abscess*, an essential preliminary to any form of drainage. Local inflammatory changes are also seen in the tubes leading from an area of bronchiectasis, but lipiodol bronchography is more useful in depicting the extent of the disease. Finally, bronchoscopy is sometimes carried out in order to obtain uncontaminated secretion for culture, and tubercle bacilli can sometimes be recovered by this means in cases where sputum cannot be obtained.

Bronchoscopy has proved to be of increasing value in the treatment of certain bronchial conditions. The recognition of the fact that *massive collapse* of the lung is frequently due to the occlusion of a bronchus by a plug of mucus or muco-pus has led to a great increase in therapeutic bronchoscopy. The blocked tube can be cleared by this means and re-expansion can be ensured. The removal of a *foreign body* is an obvious therapeutic use. Sometimes an innocent growth may be completely extirpated, although simple bronchoscopic removal is not considered to be sufficient in most cases. The treatment of malignant disease is not practicable; local removal is out of the question, and the application of radium has not been attended by good results and has led to some disasters.

Bronchoscopy is contra-indicated in cases of advanced heart disease and known aneurysm, and it should not be carried out during or shortly after severe haemoptysis. Gross sepsis in the mouth should be dealt with so far as possible before the operation is carried out.

### Thoracoscopy

The thoracoscope is an instrument constructed on the lines of the cystoscope. A *pneumothorax* must always be present before it can be introduced. The selected intercostal space is fully anaesthetized with novocaine and a large-size trocar and cannula are pushed into the pleural space. The trocar is then withdrawn and is replaced by the thoracoscope, which is an instrument equipped with a light bulb and mirror system at its distal end, and an eye-piece at its proximal end. A large part

of the pleura, both visceral and parietal, can be inspected and the relation of superficial lesions to the lung can be defined. It is even possible to remove material for section. The chief use of the thoracoscope, however, is that it enables pleural adhesions to be divided with very little disturbance of the patient. In this way an inefficient artificial pneumothorax can be made fully effective and cavities can be made to close. Without thoracoscopy a high proportion of artificial pneumothoraces would have to be abandoned because this essential closure of cavities would not be achieved.

#### Exploratory Thoracotomy

Finally, an exploratory operation may be necessary in order to settle the diagnosis. The increasing safety of operation on the chest has made it possible to examine lesions the nature of which is still doubtful, and to deal with them radically should the need arise. Exploration is invaluable in the investigation of cases of *interlobar empyema* or *lung abscess*, and in some cases of growth. It is especially valuable when early carcinoma is suspected, for it is the only means by which a decision as to the possibility of removal can be reached.

## SECTION IV

# DISEASES OF THE RESPIRATORY TRACT

### CHAPTER 13

#### THE UPPER RESPIRATORY TRACT

It is not always understood that the respiratory tract functions as a complete unit and that the state of the upper respiratory tract may play a great part in determining what occurs lower down. The examination of the mouth, throat, and nose should always be regarded as an essential part of the investigation, for which purpose it is necessary to be familiar with the local appearances in health and in disease.

#### • The Mouth

Infection sometimes descends from the mouth into the chest, although this rarely happens spontaneously. The removal of teeth and tonsils is sometimes followed by a lung abscess, and the passage of a bronchoscope may carry infected material into the bronchi. Although almost any organisms may be found in the mouth, the important ones are those which belong to the *fuso-spirochaetal* group and which are the cause of the foul sputum which is present in many cases of bronchiectasis, lung abscess, and growth. The condition of the teeth and gums must therefore be taken into account pre-operatively and in the treatment of established infections in the lower respiratory tract. The majority of the pathogenic organisms found in the mouth are sensitive to penicillin, and local administration is best carried out by means of lozenges which are allowed to dissolve slowly. In some cases the tongue shows a dark discoloration during treatment; the significance of this change is not known.

*The tonsils.*—Disease of the tonsils is not a very common antecedent to disease in the bronchi and lungs. It is necessary to realize this, for tonsillectomy is often confidently recom-

mended with a view to the relief of such conditions as recurrent bronchitis or asthma, and the operation frequently fails to achieve the desired result. Infected tonsils are often an expression of infection in the nasal sinuses and, in such cases, their removal will not effect a cure. In some cases there is no doubt that enlarged and chronically inflamed tonsils can cause an irritating cough, which is mechanical in origin and which will be relieved by their removal. The chief respiratory complication of tonsillectomy is lung abscess, and this development should be suspected in any patient who has fever, with or without respiratory symptoms, within ten days of the operation.

**Adenoids.**—These result from hypertrophy of the lymphoid tissue which is normally present in the nasopharynx in children and which is analogous to the tonsil.

The presence of adenoids leads to nasal obstruction and therefore to mouth breathing. The child snores when asleep, and the condition may be so bad as to interfere with rest. There is concurrent nasal catarrh and often a cough.

The appearance of the "adenoid child" is characteristic, for the mouth is usually open and the upper lip is often retracted. The child appears dull and stupid. Catarrh at the back of the nose may lead to defective hearing, and otitis media is not uncommon. The voice is often thick and nasal.

Descending infections of the respiratory tract are common sequels and may cause actual deformity of the chest, such as the "pigeon breast" and Harrison's sulcus. There may be associated bronchitis or asthma. The child is commonly pale and may be anaemic, the lymphatic glands in the neck are often enlarged, and the pad of adenoid tissue can be palpated. The tonsils are usually hypertrophied as well.

If left alone, adenoid tissue tends to atrophy at puberty, but by this time the seeds of disease have been sown and organic changes may have resulted. Adenoids should always be removed, together with the tonsils, when there is evidence that they are the cause of ill-health. Following operation the general health must be carefully supervised and breathing exercises should be instituted.

A *long uvula* may be the cause of a chronic unproductive cough which comes on *when the patient lies down*. Recognition of the cause of the cough is easy if the possibility is considered. Shortening of the uvula is a simple remedy.

The *pharynx* is exposed to many kinds of infection. Acute pharyngitis is nearly always associated with an acute infection in the tonsils or sinuses and causes pain and dysphagia. Chronic pharyngitis is associated with chronic infection in the tonsils or sinuses, or with excessive smoking and spirit-drinking. The mucous membrane is often red and granular, or it may appear smooth and shiny. The condition causes a chronic irritative and useless cough, and treatment is that of the cause. *Retro-pharyngeal abscess* usually occurs in children and may result from direct infection or, in chronic cases, from tuberculous caries of the underlying vertebrae. The chief symptoms are dysphagia, dyspnoea, and cough, and there may be a fixed position of the head. Diagnosis is not always easy, but it may be suspected by careful inspection and palpation, and a lateral X-ray of the cervical spine will afford confirmation. The treatment is surgical.

### The Nasal Passages

The nasal passages are divided into three by the turbinate bones. The upper passage, in the region of the ethmoid air cells, carries the smallest amount of inspired air. The two lower passages are those commonly used in respiration. The nasal mucosa consists of a transitional epithelium, the superficial layer of which is composed of ciliated columnar cells. The membrane is very vascular and is richly supplied with nerves which take their origin chiefly from the trigeminal and connect with the vagus nuclei in the medulla. There is thus an intimate connection between the mucous membrane of the nose and the innervation of the bronchi. This rich supply of nerves and vessels leads to certain parts of the membrane having the properties of erectile tissue, and irritation of the nose is very apt to be accompanied by vascular swelling in the region of the ethmoids and the middle turbinates.

Opening laterally from the nasal passages are the *nasal sinuses*. The maxillary antra, and the frontal, ethmoid, and sphenoid air cells are of little consequence in health, but they play an important part in the causation of disease in the respiratory tract as a whole. The state of the antra and frontal sinuses can be investigated by transillumination, but the remaining air cells can only be accurately visualized by means of X-rays.

"*Sensitive spots.*"—When a probe is passed into the nose and allowed to touch the nasal septum, especially in the deeper part, evidence of sensitiveness is often elicited by means of a sneeze or cough reflex; in some very sensitive patients bronchial spasm may result. This observation may be important in showing the presence of a nasal factor in cases of asthma.

*Polyp formation.*—Polypi may result from chronic infection, with hypertrophy of the mucous membrane; a similar formation is often seen in cases of "allergic" nasal mucosa. While they commonly cause purely nasal symptoms, such as recurrent catarrh and inspiratory obstruction, in some cases they are associated with attacks of asthma. Removal of polypi may be followed by cessation of asthmatic attacks, but it is also true that the removal of polypi may result in attacks of asthma in patients who have previously been free from this symptom. A great deal of experience is needed to know when to operate in these cases, and conservative measures would appear to be the safest as a rule.

*The common cold (coryza).*—Even now we know comparatively little of the causation, and still less of the treatment, of this universal affliction. There can be no doubt that it is an infection, probably with a virus, although many other organisms are found in the nasal secretion after the first day or two. That the infection is not entirely local is shown by the symptoms of general toxæmia, malaise, anorexia, and aching which so frequently cause the victim to feel ill before the local symptoms develop. It is also true that, in spite of the fact that colds are thought to be due to a virus, a stock anti-catarrh vaccine is useful in keeping the individual free from infection. Secondary infection plays an important part in determining the severity of the symptoms and the duration of the disorder and, in those who are prone to winter colds, a stock vaccine is always worth a trial. A very simple, effective method of prevention is the use of Glegg's mixture, which consists of—

|                     |   |   |                                  |
|---------------------|---|---|----------------------------------|
| R                   |   |   |                                  |
| Paraffin liq.       | . | . | 3 parts                          |
| Paraffin molle alb. | . | . | 1 part                           |
| Menthol             | . | . | $\frac{1}{2}$ grain to the ounce |

to be instilled into the nose with a pipette.

When the condition is fully developed, quinine is the drug

which appears to be the most popular and perhaps the least ineffective, although cinnamon and other aromatic oils are often recommended.

Nasal catarrh is often the first symptom of measles and, in children, the membrane of the inner surface of the cheeks should be inspected for *Koplik's spots*. A child who has been in contact with measles and who develops nasal catarrh should be isolated.

Coryza is also a symptom of sensitiveness to iodine. It is associated with conjunctivitis and a skin eruption.

*Hay fever.*—This is an allergic condition which results from hypersensitiveness to pollen. In this country it is usual to find the patient sensitive to *Timothy grass*, and in the United States to ragweed. The patient who has hay fever in this country may have no symptoms elsewhere, and conversely, a patient may have hay fever for the first time on going abroad if he happens to be sensitive to a pollen which is a prominent feature of the flora of the district.

Usually the condition shows itself in adolescence and tends to become less severe in middle age. The symptoms are characteristically seasonal. In this country they commence as a rule in May and terminate in July.

The common symptoms are sneezing and running of the nose, together with irritation of the eyes and marked inflammation of the conjunctiva. They are most prominent when the patient is in the open air in contact with the pollen, but they may come on at any time if the conditions are suitable. In addition, there is a certain amount of general disturbance and the patient may feel out of sorts and depressed. In severe cases there may be bronchial spasm (hay asthma).

The diagnosis is usually simple if the seasonal incidence is noted; there is nearly always a family history of one or other of the allergic conditions, and the patient may show other clinical evidence of the allergic state.

Confirmation should always be sought by means of the protein sensitivity tests (p. 122), which give a positive reaction to the offending pollen. The allergic nature of the condition is also shown by an increase in the number of eosinophils in the blood, and by examination of the nasal discharge, which may show a great number of eosinophil cells.

Treatment should be directed towards reducing the sensitive-

ness of the patient, and this may be done in two ways, local and general.

If there is gross disease in the nose, such as polyp formation, this should be corrected, but extensive operation is best avoided as a rule. A satisfactory method for producing local desensitization is by zinc ionization, the technique of which is quite simple.

About ten thin flat pieces of cotton-wool, between one and two inches square, are prepared and soaked in 2 per cent zinc sulphate solution, the excess fluid being squeezed out. The patient sits on a chair and the nose is carefully packed with the cotton-wool pledgets, care being taken to see that they lie flat against the mucous membrane. The first piece is inserted high up into the ethmoid region, the second along the septum, and the third and fourth along the lateral side of the nose, and the remaining space is then filled up. Except in very nervous patients, both sides of the nose may be treated simultaneously.

A rubber head-band, which contains a metal plate with two screw-holders attached, is then fixed round the head. Two curved zinc rods, the distal ends of which are wrapped in cotton-wool soaked in the zinc solution, are then fixed to the head-band and so adjusted that firm contact is made with the plugging in the nose. The hand of the patient is placed upon a moistened metal plate which is connected to the negative lead in the battery, and the positive lead completes the circuit from the battery to the metal part of the head-band. A current of between 4 and 5 milliamperes is allowed to flow for 20 minutes. The apparatus is then disconnected and the nose unpacked. Care must be taken to make certain that the correct number of pieces of cotton-wool are removed from the nose. Sometimes ribbon gauze soaked in zinc solution is used instead of cotton-wool to plug the nose. This method is perhaps a little simpler, but it is doubtful whether as good contact can be made. The usual course consists of four applications at weekly intervals and it is best to begin in mid-April. The treatment should be repeated annually for several years. The great advantage of this method is that it can be applied with success even after hay fever has actually commenced.

Zinc ionization has also been employed with success in cases of paroxysmal rhinorrhoea, and it has proved beneficial in cases of spasmodic asthma in which there is a pronounced nasal factor. It should not be attempted when there is local sepsis. The results of treatment are good in about 80 per cent of cases.

For many years active immunization has been employed with varying success. A course of subcutaneous injections of



extract of Timothy grass pollen is given for some weeks before the hay fever season begins. Many reliable preparations are on the market, so that the material is readily available. It is advisable first to test the sensitivity of the patient by means of an intracutaneous injection of the solution which is to be employed, and to commence with a dose small enough to avoid causing any marked reaction. Injections may be given at intervals of from three days to a week, according to the amount of time available. The results are not altogether dependable, and they are inferior to those obtained with zinc ionization.

Avoidance of contact with the pollen is, of course, a certain method of avoiding attacks, and with those patients who can manage it this may be done during the hay-fever season by means of a sea voyage or a journey abroad, especially to a high altitude. This is impracticable in most cases. Benadryl tablets, taken by the mouth, have recently been introduced and satisfactory results have been reported from their use. They are intended purely for the treatment of the acute symptoms.

Treatment of the attack is not very satisfactory. The nose may be sprayed at frequent intervals with a 1 in 100 solution of adrenalin, or benzedrine may be inhaled. Aestivin drops are helpful in relieving the conjunctival irritation.

*Paroxysmal rhinorrhoea.*—The symptoms are similar to those of hay fever, but the condition depends upon a vasomotor instability without pollen hypersensitiveness, and the attacks occur at any season. They result from mechanical irritation, such as the inhalation of dust, from nervous influences, or from active disease in the nose, either inflammatory or traumatic. The ocular symptoms are less prominent, but there may be associated asthma. In mild cases relief may be obtained from the use, twice daily, of a nasal spray containing ephedrine, 3 per cent, pot. sulph.,  $\frac{1}{2}$  per cent, in water, followed after five minutes by a simple alkaline nasal douche; the best consists of equal parts of sodium chloride and sodium bicarbonate, a small teaspoonful of the powder in a half tumbler of warm water. The results of zinc ionization are often dramatic, and this is the treatment of choice. Operations for ventilation of the sinuses and the removal of polypi may be undertaken, but they are not to be lightly advised, for severe asthma may follow.

*Sinusitis.*—There is a close connection between disease in

the nasal sinuses and in the lower respiratory tract. In fact, the term "broncho-sinusitis disease" has been coined in the United States in order to emphasize this relationship.

In acute sinusitis there may be local pain, according to the situation of the infected air cells; the pain will be on one side of the face if an antrum is involved, towards the eye when there is ethmoiditis, or there may be a deep-seated headache when there is inflammation in the sphenoidal sinus. Infection of the frontal cells causes local pain which may be referred over the forehead.

There is usually purulent discharge from the nose, and sometimes epistaxis. General symptoms may be prominent, especially fever and toxæmia. In some cases there is a blood-stream infection, and the possibility of sinus infection should always be kept in mind when considering the cause in obscure cases of septicaemia, pyaemia, or pyrexia of unknown origin. In these cases treatment is a very delicate matter, involving the consideration as to whether drainage should be performed, and close co-operation between the physician and surgeon is essential.

Chronic sinus infection does not always cause characteristic symptoms, yet it is almost constantly found to be present in cases of *recurrent bronchitis*, *asthma*, and *bronchiectasis*, and there can be little doubt that direct downward transfer of infected material plays an important part in aggravating, and perhaps causing, these conditions. The local symptoms are intermittent purulent nasal discharge and headache, or even mental dullness. Careful examination and transillumination are essential, and this may be supplemented by X-rays if necessary. It has been found that radical operations in cases of bronchiectasis are likely to be unsuccessful if the sinuses are grossly infected, and this point must be kept in mind when considering the advisability of lobectomy.

The operative treatment of chronic sinus infection is often unsatisfactory and the condition of the patient is even sometimes changed for the worse. When there is pus in an air cell a drainage operation should usually be undertaken, but when the mucous membrane is thickened or chronically inflamed operation does not usually help. Interference with infected sinuses is not without danger of consequent meningitis or bronchopneumonia and it is a good practice to administer a course of sulphonamide both before and after operation.

Intranasal infection can be treated with success by the local instillation of an emulsion of one of the sulphonamide group. It is as yet too early to assess the value of penicillin inhalations, although it is to be expected that this method of treatment will prove to be very useful when the infecting organism is sensitive.

Many patients who have chronic sinusitis improve with regular attention to the hygiene of the nose. Ephedrine sprays and ephedrine "replacements", and simple alkaline douches are valuable; vaccines are helpful, provided that they are given in *small doses*, starting with not more than  $\frac{1}{4}$  million organisms. In addition, considerable benefit is obtained from the local application of infra-red rays in some cases.

## CHAPTER 14

### THE LARYNX

**Foreign body.**—Foreign bodies are usually portions of food, nuts, pins, or buttons. Small foreign bodies often enter the larynx but they do not as a rule give rise to much trouble, for they are either quickly expelled or they slip through into a bronchus. The adult patient is usually aware of the accident, as there is an immediate paroxysm of cough. Should the foreign body be sharp, there may be a certain amount of bleeding but other complications are rare, although infection and acute oedema of the glottis may occur. Treatment consists in removal, preferably by direct laryngoscopy.

A large foreign body may become impacted in the larynx. In these cases the symptoms are always urgent, with choking, intense dyspnoea, and cyanosis. Urgent measures are necessary and, if it cannot be removed, immediate tracheotomy may be required. In the absence of expert assistance these cases are likely to end fatally.

**Acute laryngitis.**—This may occur at any age and in either sex.

1. It may be a primary infection, or a complication of a cold or influenza. It may also be a feature of certain specific fevers, especially *measles*, *smallpox*, and *typhoid*.
2. It may result from the inhalation of steam or irritant vapours.

The onset of acute laryngitis is abrupt, with irritation in the throat. The voice becomes husky and may be completely lost, and there is a dry cough. General symptoms are slight, the temperature is hardly raised, and the patient does not feel ill. Dyspnoea is rare, except with irritation from gassing.

The differential diagnosis is not often difficult, being chiefly from *functional aphonia* in young women. In these the vocal cords appear normal on inspection although they do not approximate, and a sound is often produced when the patient coughs.

Treatment need not be very vigorous. There is no necessity for the adult patient to be kept in bed and, in fact, many people

remain at work with a mild attack, although this is to be deprecated. The patient should be kept in a warm, moist atmosphere at a temperature of 60°. Plenty of fluid should be taken, and a mixture containing aspirin, phenacetin, and codein is helpful. Relief of local symptoms is best obtained by means of frequent inhalations, either of Friar's balsam or of—

|  |   |   |   |   |              |
|--|---|---|---|---|--------------|
| Rx   |   |   |   |   |              |
| Ol. pini   | . | . | . | . | 5 minims     |
| Mag. carb. levis                                 | . | . | . | . | 4 grains     |
| Aq.  | . | . | . | . | ad 60 minims |
| Sig. 1 teaspoonful in 1 pint of water at 150° F. |   |   |   |   |              |

Lozenges, such as troch. phenol, may be sucked.

*Oedema of the glottis.*—This is a serious condition which may be due to local or general causes.

(i) *Local causes.*

- (a) Injury, foreign body, inhalation of irritant vapours.
- (b) As a complication of acute laryngitis, diphtheria, tuberculosis, syphilis, growth.
- (c) As a complication of acute infection in the neck; Ludwig's angina, erysipelas.

(ii) *General causes.*

- (a) Angioneurotic oedema.
- (b) Hydraemic nephritis.
- (c) Congestive heart-failure.

The onset is sudden, with *urgent dyspnoea*, *stridor*, and *aphonia*. The patient rapidly becomes cyanosed and the inspiratory movements are forced. If left untreated, the condition is likely to prove fatal.

On examination the epiglottis is found to be much swollen and easily palpable. The true vocal cords are not often involved.

Treatment is a matter of urgency and *tracheotomy* is often necessary. A 10 per cent cocaine spray may relieve the urgent symptoms, and it is sometimes advised that the swollen epiglottis should be scarified. In patients subject to angioneurotic oedema a prompt injection of adrenalin may avert a severe attack.

*Chronic laryngitis.*—In these cases there is usually some predisposing cause, such as gout, alcohol, or excessive use of tobacco. The condition may be occupational in those who

habitually strain the voice, such as clergymen or street hawkers. Chronic sinus infection is often present.

The voice is hoarse and may even be lost at times. There is irritation in the throat and spasmodic cough with mucoid sputum. On direct observation the mucous membrane is seen to be swollen, the vocal cords are thick, and there is some congestion as a rule. The condition may not be easily distinguished from early tubercle, syphilis, or growth, all of which may simulate chronic laryngitis very closely in the early stages; in any doubtful case full investigation should be undertaken. The condition is chronic and the voice may be permanently impaired.

The most important indication in treatment is *rest of the voice* for a considerable period. Alcohol and tobacco should be forbidden. Inhalations of oil of pine or Friar's balsam are frequently beneficial, and a 4 per cent solution of zinc chloride may be applied locally on alternate days for two weeks. Sinus infection should receive attention.

*Tuberculous laryngitis.*—This is a condition which is always secondary to tubercle of the lung, although the laryngeal condition may be advanced while the lung condition is relatively mild and quiescent.

The inflammation commences in the posterior part of the ary-epiglottidean folds and in the arytenoid region. It spreads forwards and eventually reaches and destroys the epiglottis. The prominent symptoms are *loss of voice* and *pain on swallowing*. The onset is usually insidious, with some huskiness of the voice and laryngeal irritation. This progresses to chronic hoarseness and eventual loss of the voice. A husky cough develops, if it has not been present before, and the amount of sputum depends upon the extent of the lung lesion. Dysphagia occurs when the infection has reached the epiglottis or when ulceration has spread to the pharynx. Pain on swallowing may be extreme. Symptoms of toxæmia, fever, sweats, and loss of weight occur as the disease progresses and give an important clue to the diagnosis.

On direct examination the vocal cords are seen to be pale and thickened. Infiltration occurs early and may result in two quite different types of condition. (i) There may be granulomatous masses which resemble papillomata. (ii) In other cases typical tuberculous ulcers may be present. These

are usually pale, rather shallow, and covered with exudate, causing a "worm-caten" appearance of the cords. When the disease is more extensive there is gross loss of substance and the epiglottis may disappear entirely. Spread to the deeper tissues results in perichondritis and necrosis of the cartilages, which may occasionally be coughed up. Stenosis of the larynx is rare.

In addition to the local changes, tubercle bacilli will be found in the sputum and there will be clinical and radiographic evidence of active pulmonary tuberculosis. In cases where there is difficulty in finding the bacilli, material obtained by swabbing the ulcers should be examined.

Experience shows that the prognosis depends upon the type of laryngitis present, and very useful information can be obtained from laryngoscopy. Briefly, it may be stated that cases in which warty masses are present usually do well, whereas those in which ulcers predominate have a bad outlook.

The first essential in treatment is *complete silence*, which may need to be maintained for as long as six months. In addition the lung lesion should be treated vigorously, and great improvement in the laryngeal condition is often seen to follow the induction of a pneumothorax, or a phrenic avulsion. Local treatment consists in exposure to ultra-violet light, which appears to be very useful. The larynx may be painted with a 25 per cent lactic acid solution. A damaged epiglottis may be removed for the relief of dysphagia, but the local condition is apt to spread more rapidly after this has been done. Relief of pain may be accomplished, in part at least, by insufflating orthoform powder, gr. x, directly to the larynx, preferably half an hour before a meal. A cocaine spray is also useful, but it should be confined to cases in which the prognosis is considered to be hopeless. In the hands of a competent operator injection of the superior laryngeal nerve sometimes affords dramatic relief from pain.

When dysphagia is severe the patient should be fed on semi-solids, which are found to be more easily swallowed than either liquids or ordinary solid food. The effect of posture may be tried. The patient lies on the bed with the head well down over the side and sucks food through a tube, thus avoiding contact with the larynx during the act of swallowing (Wolfenden's posture).

*Syphilitic laryngitis*.—This is comparatively rare and may occur in several forms.

1. In *congenital syphilis* there may be catarrhal laryngitis, which is more persistent than the simple form. The diagnosis is suggested by noting the presence of other stigmata.
2. In *secondary syphilis* there may be a subacute laryngitis which tends to become chronic. Ulceration is rare but condylomata may be seen. The diagnosis is usually made by observing the other signs of secondary syphilis.
3. In *tertiary syphilis* the lesion commences at the base of the epiglottis. Gumma formation may occur and may simulate some stenosis of the larynx. Less commonly there is loss of tissue with deep ulceration; in this type secondary infection and acute oedema may complicate the picture. A diffuse gummatous infiltration of the whole larynx is sometimes seen.

The early symptoms are similar to those of chronic laryngitis, and it must be noted that dysphagia develops late and there is no pain. With stenosis of the larynx there may be stridor and dyspnoea.

Treatment is best carried out by means of injections of arsenic and bismuth at first. Potassium iodide should be given cautiously, if at all, as it is sometimes followed by acute oedema. When gross scarring occurs, dilatation by the passage of bougies may be necessary in order to keep the air-way clear. The part which will be played by penicillin in tertiary syphilis is still uncertain.

### Inspiratory Stridor in Children

Inspiratory stridor occurs fairly commonly in children, and at an age when it is not possible to obtain a direct history, so that it is necessary to bear in mind the clinical features of the various common types.

1. *Congenital laryngeal stridor*.—This has been thought to result from a congenitally large epiglottis, but there is no evidence to support this view, and it is more probable that the stridor results from spasm of the vocal cords. The condition is thus probably analogous to spasm of the pylorus and, like it, tends to pass off after a few weeks or months. It probably depends upon late myelination of the nerve fibres supplying the laryngeal muscles. The stridor is inspiratory, it is present from birth, it does not



vary, the child is not ill, and there are no complications. Complete recovery occurs within a few months.

2. *Laryngitis stridulosa*.—This results from mild infection, with spasm of the laryngeal muscles. It is common between the ages of two and four years and rarely occurs in infancy. The child suffers from slight cough for a few days, which increases until an attack of spasm occurs. Most commonly the child is awakened from sleep with difficulty in respiration, crowing inspiration, and cough. There may be signs of laryngeal obstruction and the child is frightened. There is little or no rise of temperature and the condition usually appears more serious than it really is. The spasm may last for two or three hours, after which it subsides. The patient is fairly well during the day, but the attacks recur for two or three nights. The outlook is good and recovery is always complete.

3. *Acute catarrhal laryngitis*.—This is a similar condition, but more serious. There is definite dyspnoea, the temperature is raised and there are no remissions. It is often the precursor of bronchitis or bronchopneumonia.

A very similar clinical picture is presented by *laryngeal diphtheria*. This occurs most commonly between the ages of three and five years and is nearly always secondary to an infection of the fauces. Occasionally it may occur as a primary condition. In the early stages there are hoarseness, harsh cough, stridor, and dyspnoea. In the milder cases there is slight membrane formation, the dyspnoea occurs in paroxysms as a result of spasm of the glottis and, as there is little obstruction, the illness is not serious. In the more serious form the stridor and dyspnoea become progressively more severe, without remissions, cyanosis increases, and the patient becomes urgently ill. This type of case is associated with abundant membrane which often invades the trachea and even the bronchi. Pulmonary collapse, pneumonia, or heart-failure may supervene. In all cases of doubt it is advisable to take a swab from the larynx for culture.

4. *Laryngismus stridulus*.—This is associated with rickets. It does not occur before the age of six months and very rarely after the age of three years. The stridor results from spasm of the muscles of the larynx and may be associated with tetany. Attacks occur most commonly at night or in the early morning, and may be brought about by any irritation. There is no cough nor alteration of the voice and the attack begins with sudden cessation of respiration, during which the patient struggles for breath and becomes slightly cyanosed. Eventually the spasm relaxes and the attack terminates with crowing inspiration. The diagnosis is made in the majority of instances by noting the signs of rickets and evidence of latent tetany. The condition is not serious as a rule and fatalities are rare.

5. *Whooping-cough*.—This is discussed in another section (p. 111).

6. *Foreign body*.—The inhalation of a small foreign body may not be noticed and it may remain in the larynx.

7. *Papilloma of the larynx*.—This is a condition which is sometimes seen in children. The stridor is continuous and the voice is affected. The diagnosis can only be made by the laryngoscopy.

8. *Tonsils and adenoids*.—In the presence of an upper respiratory infection with laryngeal catarrh there may be some degree of stridor.

9. *Thymic stridor*.—Enlargement of the thymus gland is said to cause stridor (Kopp's asthma). An X-ray of the chest may not reveal the condition and the diagnosis is then reached by a rather unsatisfactory process of exclusion (p. 294).

The term "croup" is often employed, especially by the mothers of young children, to denote an illness. The expression appears to be applied indifferently to any of the conditions characterized by inspiratory stridor and it does not refer to any specific disease. In most cases it may be presumed that the term implies acute catarrhal laryngitis.

The treatment of stridor is partly routine, but it also depends upon the cause. In congenital laryngeal stridor reassurance of the parents is all that is necessary. For laryngeal catarrh a steam kettle or a hot bath is very helpful, and an emetic, *Tr. ipecac.*, 40–60 minims, will hasten the end of the attack. The child should be kept in a warm well-ventilated room. Acute laryngitis is treated in the same way, and in addition a useful medicine is—

|                        |   |   |   |   |               |
|------------------------|---|---|---|---|---------------|
| R.                     |   |   |   |   |               |
| <i>Tr. belladonnae</i> | . | . | . | . | 5 minims      |
| <i>Tr. ipecac.</i>     | . | . | . | . | 3 minims      |
| <i>Syr. tolu.</i>      | . | . | . | . | 30 minims     |
| <i>Aquam</i>           | . | . | . | . | ad 120 minims |
| Four-hourly.           | . | . | . | . |               |

Should there be a strong suspicion of the possibility of diphtheria, it is wise to give antitoxin in full doses as early as possible; in this type of case symptoms of progressive obstruction may indicate the need for tracheotomy. The treatment of laryngismus stridulus is usually the treatment of rickets; the condition is less commonly seen since rickets has become comparatively rare. The treatment of the attack is to apply heat externally to the neck for relief of spasm, or to douche the face with cold water. Severe attacks may necessitate a mustard bath, and the spasm may only be relieved when a little chloroform is given.

## CHAPTER 15

### THE TRACHEA AND BRONCHI

**DISEASE** rarely affects the trachea alone. In most infections the whole tree is involved and a composite clinical picture results.

A foreign body which is sufficiently small to pass the entrance to the larynx is too small to be retained in the trachea, and it will not be arrested until it has entered a bronchus. Injury of any sort is rare.

Acute tracheitis is present in most cases of acute bronchitis and is responsible for the raw sensation under the sternum which is a constant symptom. In *influenza* the mucous membrane of the trachea is usually intensely engorged. Drugs are of no value in relieving the symptoms of acute tracheitis, but the inhalation of soothing vapours, especially oil of pine, is helpful. Chronic inflammation of any kind is rare.

Compression and displacement of the trachea are often well shown on a plain X-ray (fig. 53).

*Displacement* of the trachea is an important sign of chest disease for it indicates the position of the superior mediastinum. The significance has already been considered (p. 39). Tumours of the trachea are uncommon. Fibroma, myoma, and adenoma are sometimes found and carcinoma may occur just above the bifurcation. When a tumour is small there will not be any symptoms, but, as it enlarges, inspiratory obstruction with stridor will result. Haemoptysis may occur with malignant growth. An exact diagnosis can only be made by endoscopy.

*Pressure on the upper part of the trachea* results from enlargement of the thyroid gland, in Graves' disease and in carcinoma. When the lower part is compressed the lesion is in the superior mediastinum and is likely to be *malignant growth, lymphadenoma, aneurysm, or a retrosternal goitre*. The symptoms are *stridor, dyspnoea*, and brassy cough. *Haemoptysis* will only occur when the mucous membrane is eroded.

# THE TRACHEA AND BRONCHI

109

## Diseases of the Bronchi

### Foreign Body

Foreign bodies may be found in the bronchi at any age. In small children it is not uncommon to find that a pea-nut has been inhaled. In addition, safety-pins, teeth, or particles of food may find their way through the larynx. The foreign body most commonly lodges in the right lower bronchus. The symptoms may be comparatively slight; a choking attack with spasmodic cough may be the only evidence, and when the foreign body is securely lodged there may be collapse of a lobe with no further symptoms for months or even years. If the foreign body has a rough surface, there may be haemoptysis. It might be expected that pulmonary suppuration would be an invariable sequel, but this is not necessarily so. Vegetable matter always causes suppuration. In cases where infection supervenes the clinical picture is that of lung abscess or of bronchiectasis.

A diagnosis may be made by X-rays when the foreign body is opaque, but many substances do not cast a shadow and therefore a negative X-ray does not exclude the possibility of foreign body. A sure method of diagnosis is bronchoscopy.

Treatment consists in removal through a bronchoscope as a rule. When the foreign body is discovered accidentally, and when no symptoms are present, it may be justifiable to await events, although it must be realized that infection may occur at any time; many cases are on record of the spontaneous expulsion of a foreign body many months, or even years, after it had been inhaled. There is no evidence that the chronic irritation caused by the presence of a foreign body ever leads to the development of malignant disease. Foreign body in a small bronchus is out of reach of the bronchoscope and, should removal be considered necessary, an open operation must be undertaken.

### Acute Bronchitis

- Acute bronchitis may occur in the following circumstances:—
1. As a specific infection.
  2. As a descending infection from the nasal sinuses or throat.
  3. As a part of the clinical picture in certain specific fevers notably in measles and in typhoid.
  4. As the result of the inhalation of irritant gases.

It is commonest in winter and spring and appears to attack those whose resistance is lowered.

In the average adult, acute bronchitis is a comparatively mild disorder, but in infants and in old people it may be extremely serious and may be a terminal event.

In mild cases there is little constitutional disturbance, the temperature is usually less than 100°, and there is slight malaise. The earliest local symptom is a raw sensation under the sternum, with cough and scanty viscid sputum. When cough is severe the sputum may contain streaks of blood, but this is not common and should always raise doubt about the diagnosis. There is no pain as a rule and little shortness of breath. After two or three days the sputum becomes more copious and mucopurulent in character. Resolution proceeds gradually for a week or more.

On physical examination the patient may be mildly toxic, with a slightly raised pulse rate. The respiration rate is not as a rule affected in adults. In infants the respirations are raised, but usually not above 40 per minute. This point is of value in distinguishing bronchitis from broncho-pneumonia, in which the respirations are considerably higher.

The only physical sign in the chest is the presence of bilateral *rhonchi* and *sibili*, indicative of sticky secretion in the bronchial tubes.

Pathological investigations are not usually done, but in cases where the bronchitis fails to resolve properly, or where there are repeated attacks for no apparent cause, the sputum must be carefully examined for tubercle bacilli. An X-ray will not show any changes in acute bronchitis, but will be of great value in revealing incipient lung disease. The bacteriology of the sputum is not usually helpful. The organisms commonly found are *Mic. catarrhalis*, *pneumococci*, *streptococci*, and *H. influenzae*, and more than one kind may be present.

*Treatment.*—The patient should remain in a warm room for two or three days, although it is not necessary to advise complete rest in bed as a rule. Diet should be light, and plenty of fluid should be taken. A hot bath at night, together with 10 grains each of Dover's powder and aspirin, is useful in promoting sleep and in encouraging diaphoresis. In the early stages, a mixture may be prescribed in order to loosen the phlegm. A valuable prescription is—

# THE TRACHEA AND BRONCHI

111

|    |                    |            |
|----|--------------------|------------|
| R. | Sod. bicarb.       | 15 grains  |
|    | Tinct. ipecac.     | 10 minims  |
|    | Tinct. opii camph. | 20 minims  |
|    | Syr. tolu.         | 30 minims  |
|    | Aq.                | ad 1 ounce |

Penicillin, both by inhalation and by injection, is indicated when the attack is at all severe, provided that the sputum contains a preponderance of sensitive organisms.

When the effort of coughing is out of proportion to the result a simple linctus, such as Geo's linctus (p. 116), may be given. Inhalations are useful for substernal discomfort; Friar's balsam or oil of pine (p. 102) are often prescribed. It is doubtful whether chest liniments have any real value, but a camphor liniment is sometimes advised, especially in children. Other local applications are not necessary.

## Whooping-Cough

Whooping-cough is a specific fever due to infection of the trachea and bronchi with *H. pertussis*. It is commonest in young children, although it may occur in adults, and even in the aged, in whom it may be a serious disease. The incubation period is about two weeks.

The cause of the characteristic whoop is not known. The mediastinal glands are always enlarged and inflamed, and it has been suggested that pressure by these glands on the lower part of the trachea is responsible for the paroxysms.

This disease is very infectious in the early catarrhal stage, before the diagnosis is usually made; isolation is therefore often advised too late to prevent the development of contact cases.

The clinical condition may be described in three stages, catarrhal, paroxysmal, and resolution.

During the first week the patient develops mild fever and a cough of increasing severity and of a peculiar character, in that it consists of a series of short, sharp, expiratory barks. The occurrence of this type of cough should always raise the suspicion that the condition is whooping-cough, even before the characteristic whoop makes its appearance. There are two other helpful clinical points at this stage. The cough is often sufficiently severe to cause vomiting, and there may be a

characteristic sublingual ulcer. This only occurs in infants in whom the lower central incisor teeth are prominent, and is caused by the pressure of the under surface of the tongue against the sharp margins of the teeth (Riga's ulcer).

During the second week the characteristic whoop makes its appearance. After intensive expiratory efforts the laryngeal spasm suddenly relaxes and air is violently sucked in through the glottis. At this stage there can be no mistake in the diagnosis. On examination of the chest there may be very few physical signs, although rhonchi and sibilii are sometimes heard. In severe cases there may be considerable venous congestion, especially of the face, during the paroxysms. Even at this stage the temperature is not much raised in the absence of complications. The third stage is one of defervescence, in which the cough gradually diminishes in severity and the patient is clinically well; but it must be noted that the whoop may persist for a very considerable time and is apt to recur for months, or even for one or two years, if the patient develops a subsequent respiratory infection.

*Early diagnosis.*—Even before the whoop appears the type of cough should arouse suspicion. Confirmation can be obtained at a very early stage by making the patient cough on to a culture plate held in front of the mouth for about fifteen seconds. Incubation will reveal the causal organism. A pronounced lymphocytosis is also very suggestive.

The complications of whooping-cough may be either infective, mechanical, or vascular.

The most important infective complication is *bronchopneumonia*, which is fairly common. The combination of whooping-cough with bronchopneumonia makes the prognosis more grave than in simple bronchopneumonia. The signs in the chest are those of patchy consolidation, and any of the complications of pneumonia may develop.

Mechanical complications result from distension of the alveoli as a result of the strain of coughing. Acute vesicular emphysema (p. 162) is common and some permanent damage may be left; interstitial emphysema is rare. Spontaneous pneumothorax may occur. Weakening of the bronchial wall may be a factor in causing subsequent bronchiectasis.

The strain of coughing tends to embarrass the right side of the heart and consequently the venous circulation. Right

## THE TRACHEA AND BRONCHI

113

heart strain with dilatation of the right ventricle is not uncommon, but recovery is usually complete. In severe cases heart-failure may result. Haemorrhages may occur in various parts, notably in the conjunctiva, the brain, and the skin. *Haemoptysis* in small quantities is not uncommon. The most important sequel is bronchiectasis. The inflammation in the mediastinal glands may sometimes activate a latent focus of tuberculosis.

*Treatment.*—The milder cases need not be kept in bed, so long as the temperature is not raised and there are no serious complications. Plenty of fresh air is essential. The diet should preferably be semi-solid, as the more solid foods tend to set up spasms of cough. When vomiting occurs after food the patient should be fed again immediately in order to avoid malnutrition. The cough can usually be controlled by bromide and belladonna, and it must be remembered that children stand the latter drug very well. When the spasms are difficult to control ephedrine may be prescribed in full doses, or ether in olive oil may be given per rectum. The stronger cough sedatives, containing opium or codein, should not be given to children, but they are frequently proscribed for adults. When the whoop is persistent small doses of X-rays directed to the mediastinal glands may be effective.

Vaccines made from *H. pertussis* are of little value in treatment, but they may be useful in preventing the disease if given to children who have been in contact.

### *Chronic Bronchitis*

"Chronic bronchitis" is really a degeneration of the mucous membrane, although the effects of infection are often superimposed. It is common in middle age, affects males more than females, and tends to run in families. In most cases the disease comes on insidiously, with winter cough which becomes gradually more severe each year; rarely the story is of repeated attacks of acute bronchitis leading to winter cough. Environment is to some extent concerned, and it occurs in those who are engaged in dusty occupations or who are exposed to damp atmosphere and sudden changes of temperature. It is also a sequel of mechanical irritation of the bronchi, and gassing. It is associated with the common chronic degenerative diseases of middle life, arteriosclerosis, heart disease, and gout, and



conjunction is so close as to give rise to the belief that *chronic bronchitis is primarily due to an underlying vascular degeneration rather than to infection*. This conception is of the utmost importance for it accounts for the intractable nature of the complaint and it indicates that treatment should be general rather than local.

The condition is usually associated with emphysema (p. 159), and some of the symptoms, such as dyspnoea, are referable to this. The symptoms are worse in the winter.

*Cough* is perhaps the most prominent symptom ; it is often worse at night, keeping the patient awake. It may be paroxysmal and exhausting. The sputum varies in quantity. It may be scanty and difficult to expel, or there may be a moderate amount of mucoid or mucopurulent phlegm. It is rarely copious in the absence of bronchial dilatation. In mild cases most of the sputum is coughed up in the early morning. *Wheeze* is often associated with the cough.

*Haemoptysis*, usually in the form of streaks of blood, is not uncommon. There can be no doubt about this fact, but it must be remembered that it is never safe to assume that haemoptysis is due to chronic bronchitis until the other, more serious, causes, such as tuberculosis, bronchiectasis, and growth, have been excluded.

*Shortness of breath* is very common. This results, not from chronic bronchitis, but from associated emphysema ; sometimes it is cardiac in origin.

On physical examination the patient may be either fat or thin. The chest nearly always shows evidence of emphysema, and the only sign of the bronchitis is *rhonchi* and *sibilii*, which are not always present.

The investigations which should be performed as a routine are :—

The sputum should be examined bacteriologically, and search should be made for tubercle bacilli.

An X-ray of the chest should always be taken. In the typical case there will be evidence of emphysema, with some increased striation as a rule. The right side of the heart is usually somewhat enlarged and the pulmonary cone is prominent. It is only necessary to do a lipiodol examination if the presence of copious purulent sputum suggests that there may be bronchiectasis, or if there should be reason to suspect an early carcinoma.

Chronic bronchitis is a comparatively benign condition

which lasts for many years. Most commonly, progressive emphysema leads to heart-failure, and death occurs from this or from some associated vascular lesion. The resistance to acute lung infections is diminished.

There are few complications of chronic bronchitis. Occasionally a pneumothorax may occur as the result of rupture of an emphysematous bulla. In such cases dyspnoea is usually extreme. There may be a mild cylindrical dilatation of the basal bronchi. Severe cough sometimes causes fracture of a rib.

*Treatment.*—Tobacco smoke, particularly from cigarettes, is particularly pernicious, in chronic bronchitis and relief of the cough is rarely achieved while the patient continues to smoke. The first step is therefore to advise the patient to abandon the use of tobacco.

It is desirable whenever possible to spend the winter in a mild climate, the South Coast resorts, Egypt, and California being perhaps the most suitable. The atmosphere should not be too dry and dust should be avoided; sudden changes of temperature are harmful. High altitudes must be avoided when any appreciable degree of emphysema is present. Respiratory infection aggravates the symptoms and it is therefore a good plan to give a course of vaccine, starting in September and keeping up the injections during the whole of the winter; an autogenous vaccine is preferable, and it must be remembered that the initial dose should always be very small, not exceeding  $\frac{1}{2}$  million organisms.

Medicinal treatment can be very helpful. A useful morning draught is—

|                |   |   |   |   |                        |
|----------------|---|---|---|---|------------------------|
| R <sub>x</sub> |   |   |   |   |                        |
| Sod. bic.      | . | . | . | . | 15 grains              |
| Sod. chlor.    | . | . | . | . | 5 grains               |
| Spt. chlorof.  | . | . | . | . | 5 minims               |
| Aq. anisi.     | . | . | . | . | ad $\frac{1}{2}$ ounce |

In an equal quantity of warm water.

Cough lozenges are beneficial, and the best are troc. sod. bic. (B.P.), troch. phenol. (B.P.), or

|                   |   |   |   |   |                     |
|-------------------|---|---|---|---|---------------------|
| R <sub>x</sub>    |   |   |   |   |                     |
| Ext. glycyrrhizae | . | . | . | . | 3 grains            |
| Ol. anisi         | . | . | . | . | $\frac{1}{2}$ minim |
| Troch. acaciae    | . | . | . | . | 10 grains           |

A linctus is also very helpful in relieving useless cough. A classical one is Gee's linctus :—

|                |                 |                        |
|----------------|-----------------|------------------------|
| R <sub>x</sub> | Tr. opii camph. | } equal parts of each. |
|                | Oxymel scillae  |                        |
|                | Syr. tolut.     |                        |

One teaspoonful to be taken occasionally.

Stock medicines are not of great value in the average case, but a long course of pot. iod., given in small doses, is often beneficial.

When there is evidence of bronchial spasm, as shown by marked expiratory wheeze, belladonna or lobelia may be added to the mixture in a dose of 5 to 10 minims. Senega and ammonium carbonate are of little value.

When irritating cough is a prominent feature, inhalations may be prescribed. The simplest method is to inhale the steam from boiling water to which 1 dram of tinct. benzoin co. has been added, or menthol and oil of pine may be used (p. 102).

When emphysema is advanced, it is necessary to be cautious in the prescription of sedatives which contain morphia or heroin. The barbiturates are safer and should be used for promoting sleep.

*Tuberculous ulceration* of the bronchi has been revealed through the bronchoscope, on many occasions and it is no longer correct to describe this as a rare condition. In addition to ulceration, granulation tissue and strictures are sometimes seen. It is wise to suspect the possible presence of tuberculous ulceration in a bronchus if the sputum is regularly reported to contain tubercle bacilli when the X-ray appearances in the lung would suggest that the bacilli should have disappeared. The condition is difficult to treat, and the best method is to apply a 30 per cent solution of silver nitrate to the ulcers through a bronchoscope. A word of caution is necessary about bronchoscopy in cases where the sputum contains tubercle bacilli. Incautious handling of the instrument may result in spread of the disease to hitherto unaffected parts of the lung.

*Syphilis* may occasionally produce a localized gummatous infiltration in a large bronchus. The process may result in an inflammatory stricture with pulmonary collapse. This condition can only be distinguished from a growth by bronchoscopy and microscopic section. A positive Wassermann reaction is not, by itself, sufficient evidence. In cases of doubt an intensive course of anti-syphilitic treatment should be given, and the result considered before a final diagnosis is made.

## CHAPTER 16

### ASTHMA

THE term "asthma" signifies "*laboured breathing*". In this sense it is sometimes employed to describe the dyspnoea of chronic heart-failure or of uraemia, but it is most frequently used to denote a common symptom-complex, *spasmodic asthma*. As the name implies, spasmodic asthma is not a specific disease but rather a *unique symptom produced by the abnormal response of the respiratory centre to a stimulus*.

The attack consists of a spasm of the plain muscle of the bronchi, together with some swelling of the mucous membrane. The effects are those of *vagus over-action*, and they can be counteracted by stimulation of the sympathetic. Many stimuli are capable of provoking this response *when conditions in the centre are favourable*, and each patient who complains of asthma must therefore be thoroughly overhauled in order to discover what potential exciting causes are present.

The stimuli which cause the attack may be divided into six groups, and it must be understood that more than one factor is often present in a particular case, so that *complete investigation* is always necessary. Further, the most obvious factor found is not necessarily that which is usually responsible for initiating the attack. Treatment of an apparently minor factor is often sufficient completely to relieve the symptom, although the *tendency* to asthma may remain. The principal factors are found to be: (1) allergic, (2) upper respiratory, (3) broncho-pulmonary, (4) alimentary, (5) endocrine, and (6) psychological. The condition may occur in either sex and may commence at any age.

1. *The allergic factor*.—The term "allergy" implies a state of inherited hypersensitiveness to a foreign substance which is usually, although not invariably, a protein. Acquired sensitiveness to a foreign protein is rare. Bakers sometimes become sensitive to flour and develop asthma in consequence. Dust sensitiveness may be due to the protein content of the dust with which the patient comes in contact, or it may be a pure mechanical effect on the mucous membrane of the nose.

The assessment of the factor depends on the following points. In the allergic individual asthma generally starts in childhood or in early adult life; there may be a history of other allergic conditions, such as infantile eczema, urticaria, hay fever, or migraine; the family history usually affords instances of one or more allergic conditions in relatives; the blood may show an eosinophilia, and it may be possible to demonstrate sensitiveness to one or more proteins by means of skin tests.

The interpretation of the skin tests is not always easy. Although the patient may be demonstrably sensitive to some common protein, it does not always follow that attacks of asthma will occur whenever the patient is brought into contact with the offending substance. This is particularly well shown by patients who are sensitive to feathers. Many of them do not actually have attacks of asthma on every occasion on which they sleep on a feather pillow, but attacks are prone to occur when some other factor, such as nasal catarrh, dyspepsia, or worry, lowers their resistance. Patients may of course be sensitive to substances other than those included in the test groups. Many asthmatics are sensitive to drugs, especially aspirin, and care should always be exercised in prescribing for the allergic patient.

The presence of an allergic factor shows a *tendency* to asthma. It may be looked upon as indicating that the patient is *liable to develop an attack of asthma* whereas other, non-allergic, subjects might respond to the same stimulus by producing some quite different symptom.

2. *The upper respiratory factor.*—This is very commonly present in asthmatics of every age. On taking the history various points may be noted. A history of hay fever, so long as it is the genuine allergic condition, is simply evidence of the presence of an allergic factor. Frequently, however, patients complain of a syndrome, very similar to hay fever, but not confined to the proper season. In these cases skin tests are negative and the condition is a *vasomotor neurosis*. It is a well-recognized associated condition, which frequently acts as the exciting stimulus. In such cases the nasal mucous membrane may look normal or pale, although, if the patient is seen in an attack, the membrane may look red and congested. More gross organic disease is sometimes present in the nose. Polypi may be discovered on examination, or it may be found that one or more nasal sinuses are infected. Inspection of the nose is of

great importance. There may be considerable hypertrophy of the turbinate bones, or the nasal septum may be grossly deflected, thus interfering with free ventilation. It is sometimes noted that, on passing a probe lightly over the nasal septum, one or more "sensitive spots" can be detected, as shown by an immediate sneeze or cough reflex elicited when the spot is touched.

The relation of the tonsils to asthma is more difficult, for, although they are frequently found to be infected, their removal does not often improve the condition.

In assessing the nasal factor the history is at least as important as the local examination. Patients in whom nasal trouble definitely acts as the exciting factor nearly always give a history of recurrent nasal catarrh or sneezing attacks immediately before the attack of asthma begins, and there is often a history that contact with dust excites the attack.

3. *The broncho-pulmonary factor.*—The chest must be carefully investigated from the point of view of possible organic disease. In a small proportion of cases there is active *pulmonary tuberculosis*; the sputum must always be examined and an X-ray should be taken. In other cases there is chronic bronchitis, or some degree of bronchiectasis, and a bronchogram may be necessary. Less commonly there may be extensive fibrosis in the lungs, either as a result of past inflammatory disease, or with silicosis. In chronic cases there is often a considerable degree of emphysema.

Apart from the question of organic disease in the lungs themselves, the respiratory movements are worthy of careful study. It is commonly found that the asthmatic patient makes very little use of the lower intercostal muscles in breathing, and the diaphragm is frequently low in such cases. The study of respiratory movements should be carried out both by direct observation of the patient and also on the X-ray screen. A double-exposure film (p. 69) may be helpful.

4. *The alimentary factor.*—This is common in patients who suffer from nocturnal asthma. Attacks are apt to follow heavy meals, especially if taken late at night. In these cases there may be no sensitiveness to any particular article of food, and it is more a question of gastric distension acting as an exciting stimulus. There is usually an atonic dyspepsia and on investigation it is often found that there is a *complete absence of hydro-*

*chloric acid in the gastric juice.* Constipation is sometimes a factor. In every case it is essential to remedy the digestive defect if the condition is to be relieved.

5. *The endocrine factor.*—The gland most intimately concerned is the suprarenal, as is shown by the instant effect of injections of adrenalin in the attack. Injections of pituitrin have a similar effect in many cases, and therefore the pituitary would appear to be concerned to some extent. The connection with the thyroid gland is not very obvious, yet many asthmatics are thin and have tachycardia, and there may even be mild exophthalmos; occasionally the asthmatic patient resembles a mild case of hyperthyroidism. There is also a connection with the gonads, especially in the female. It is quite common to find that the attacks of asthma are much more marked just before a period is due to start, and it is also frequently noted that asthma is completely relieved for the time being by pregnancy. In other cases, however, the asthma may date from a pregnancy.

6. *The psychological factor.*—This factor is at least as important as any other, yet it is by far the most difficult to assess. There can be no doubt that asthma is much more likely to occur when the patient is upset or worried in any way, and the environment must always be noted. Family disturbances and jealousies, unsuitable employment, and uncongenial surroundings are factors which it is sometimes possible to overcome, with great benefit to the sufferer. The psychological factor is important from other points of view. In the first place, it is possible that an *asthmatic habit* may be contracted by contact with, or observation of, another asthmatic subject. Secondly, the presence of a psychological factor renders it very difficult to assess the results of treatment. Anything which is done which is sufficiently impressive, such as a course of injections or treatment of the nose, is likely to meet with temporary success which is not entirely due to the intrinsic merit of the treatment. Confidence in the physician is an important factor in the successful treatment of asthma.

The onset of the attack is usually sudden; there is a feeling of constriction in the chest and intense shortness of breath. The patient feels as if it is impossible to expel air from the chest and consequently takes in short gasps of air, but spends most

of his time trying to empty the chest. An upright posture is usually adopted, for, in this position, the action of the diaphragm is assisted by the weight of the abdominal contents. There may be considerable useless cough, and a little clear mucus, possibly containing threads, may be voided at the end of the attack. A few streaks of blood may appear when the cough is severe.

On examination an audible expiratory wheeze is constantly present. The chest is fully expanded, the breath sounds are distant, expiration is prolonged, and *rhonchi* and *sibilli* are heard. The temperature is not raised.

When the spasm is long continued, as in "status asthmaticus", cyanosis and sweating may occur, and even acute heart-failure, although this is comparatively rare.

Between the attacks the patient may be perfectly well, or there may be a tendency to wheeziness. In fact, *wheeze* is the most characteristic feature of asthma.

There are few complications. Spontaneous pneumothorax may occur in the attack, and emphysema, with or without bronchitis, may result when the condition is chronic. The repeated strain on the pulmonary circulation may lead, in the course of time, to right heart-failure. A bronchus sometimes becomes obstructed by sticky mucus, with consequent deflation of a lobe or of a lung (p. 167).

The diagnosis of spasmodic asthma is easy, especially if an attack can be observed. Care must be taken not to confuse the expiratory wheeze of asthma with the inspiratory stridor of tracheal compression. The symptoms of bronchitis are more constant, there is pyrexia and mucopurulent sputum, but the conditions may coexist. The "asthma" of chronic heart or kidney disease occurs for the first time in patients of middle age, in whom the primary condition should be obvious on examination.

The investigation of the asthmatic subject should be carried out as follows:—

1. *History*.—Questions are first directed to the allergic factor and concern age of onset, other allergic conditions such as infantile eczema, urticaria, hay fever, together with inquiry as to the occurrence of similar conditions in other members of the family. The state of the nose is then considered with reference to catarrh, paroxysmal rhinorrhoea, and evidence of nasal obstruction. The



symptomatology of chest disease, comprising the usual series of questions, is then gone through. It should be noted that in pure asthma there is little sputum. Haemoptysis should always be regarded with suspicion. Inquiry is next made about the digestion and the relation of the attacks to meals. Appropriate questions are then asked as to the possible connection between the endocrine system and the asthma. It is usually possible to gain a fairly clear idea, while taking the history, of the importance of the psychological factor, for the patient's own assessment is not reliable. Information on this point may also be gained from the relations.

2. *Physical examination* comes next. An ordinary routine examination of the chest is first performed in order to exclude the presence of organic disease. The degree of emphysema is noted, for this observation is helpful in prognosis. The right side of the heart is sometimes enlarged and the blood pressure is often found to be low. Abdominal examination is carried out as a routine. The reflexes convey an idea of the state of the nervous system, and tremor suggests the presence of a thyroid factor.

The examination of the nose and throat must always be carefully carried out. In the first place, direct investigation will show the state of the mucous membrane, the presence of enlarged turbinates, and polypi. The septum is then gently explored with a probe, when a cough or sneeze response indicates the presence of "sensitive spots". The throat should next be examined and finally the sinuses should be transilluminated.

The following investigations must next be carried out :—

(a) The urine is usually normal. Special tests may demonstrate the presence of a proteose, the significance of which is not understood.

(b) The sputum must be examined. It is usually scanty, clear, and viscid. Under the microscope, eosinophil cells, Curschmann's spirals, and Charcot-Leyden crystals may be seen. There are few organisms as a rule. In cases where the sputum is more copious and yellow, tubercle bacilli should be sought. When there is evidence of bronchial infection, full bacteriological examination should be made in order that a vaccine may be prepared if necessary.

(c) *Skin sensitivity tests* should be performed. It is customary to use stock proteins and it is now usual to employ in addition an extract of house dust. Protein sensitivity tests may be carried out in two ways, either by intradermal injections or by a simple scratch on the skin. Intradermal injections are more troublesome, for they need the provision of sterile liquid extracts of the substances to be tested. It also means a considerable number of injections, which are not well tolerated by many children, and a fairly high proportion of pseudo- and doubtful reactions are encountered. The scratch method is quite adequate for all ordinary cases and is much simpler to perform.

Small tubes which contain the protein to be tested in a semi-liquid base are readily obtained, and a selection is made to suit the needs of the individual case. In patients with asthma it is wise to test the sensitivity to all the common proteins, i.e. to foodstuffs, epidermals, and pollens. In cases of hay fever it is usually sufficient to test the response to pollens. In all cases it is as well to include orris root, the usual basis of face powder, among the substances to be tested. Additional substances may be used if there should be any particular indication.

The skin of the forearm is cleaned with ether or spirit and a series of compartments are drawn with a skin pencil. A drop of the material is then expressed from each tube and a small scratch, about 2 mm. in length, is made through each drop. A little practice is required to ensure uniform results, the correct depth being that which produces a faint streak of blood, showing that the skin has just been penetrated. The solutions are allowed to remain in contact with the scratch for two or three minutes and the excess is then wiped off. The results are read in twenty minutes. A positive result is indicated by the presence of a wheal, together with an area of surrounding redness. As a rule the contrast between a positive and a negative result is so clear that there is no doubt; when a result is indefinite, it should be classed as negative. In ordinary cases, when many tests are being made simultaneously, it is not necessary to use a control solution, for the contrast between the positive and negative reactions indicates the results sufficiently clearly. When only one or two tests are being made a control solution should always be employed. The swelling persists for a few hours and all traces should have disappeared in three or four days.

(d) Blood examination may reveal an eosinophilia when an allergic factor is present.

(e) An X-ray of the chest should always be taken to determine the presence of organic disease. Should there be any suggestion of bronchiectasis, a *lipiodol* examination may be done. The information to be gained from screening and from double-exposure films have been considered (pp. 52, 69).

(f) A fractional test meal should be carried out as a routine. In about one-third of all cases it will be found that there is little or no secretion of hydrochloric acid.

(g) In certain cases, when a thyroid factor is suspected, the basal metabolism may be estimated.

*Treatment.*—This must be selected for the individual patient, and it depends upon the accurate assessment of the factors concerned. It may be stated that, of all the various lines of treatment at our disposal, efforts at desensitization are the least effective. It happens that, in most cases, the presence of

symptomatology of chest disease, comprising the usual series of questions, is then gone through. It should be noted that in pure asthma there is little sputum. Haemoptysis should always be regarded with suspicion. Inquiry is next made about the digestion and the relation of the attacks to meals. Appropriate questions are then asked as to the possible connection between the endocrine system and the asthma. It is usually possible to gain a fairly clear idea, while taking the history, of the importance of the psychological factor, for the patient's own assessment is not reliable. Information on this point may also be gained from the relations.

2. *Physical examination* comes next. An ordinary routine examination of the chest is first performed in order to exclude the presence of organic disease. The degree of emphysema is noted, for this observation is helpful in prognosis. The right side of the heart is sometimes enlarged and the blood pressure is often found to be low. Abdominal examination is carried out as a routine. The reflexes convey an idea of the state of the nervous system, and tremor suggests the presence of a thyroid factor.

The examination of the nose and throat must always be carefully carried out. In the first place, direct investigation will show the state of the mucous membrane, the presence of enlarged turbinates, and polypi. The septum is then gently explored with a probe, when a cough or sneeze response indicates the presence of "sensitive spots". The throat should next be examined and finally the sinuses should be transilluminated.

The following investigations must next be carried out :—

(a) The *urine* is usually normal. Special tests may demonstrate the presence of a proteose, the significance of which is not understood.

(b) The *sputum* must be examined. It is usually scanty, clear, and viscid. Under the microscope, eosinophil cells, Curschmann's spirals, and Charcot-Leyden crystals may be seen. There are few organisms as a rule. In cases where the sputum is more copious and yellow, tubercle bacilli should be sought. When there is evidence of bronchial infection, full bacteriological examination should be made in order that a vaccine may be prepared if necessary.

(c) *Skin sensitivity tests* should be performed. It is customary to use stock proteins and it is now usual to employ in addition an extract of house dust. Protein sensitivity tests may be carried out in two ways, either by intradermal injections or by a simple scratch on the skin. Intradermal injections are more troublesome, for they need the provision of sterile liquid extracts of the substances to be tested. It also means a considerable number of injections, which are not well tolerated by many children, and a fairly high proportion of pseudo- and doubtful reactions are encountered. The scratch method is quite adequate for all ordinary cases and is much simpler to perform.

Small tubes which contain the protein to be tested in a semi-liquid base are readily obtained, and a selection is made to suit the needs of the individual case. In patients with asthma it is wise to test the sensitivity to all the common proteins, i.e. to foodstuffs, epidermals, and pollens. In cases of hay fever it is usually sufficient to test the response to pollens. In all cases it is as well to include orris root, the usual basis of face powder, among the substances to be tested. Additional substances may be used if there should be any particular indication.

The skin of the forearm is cleaned with ether or spirit and a series of compartments are drawn with a skin pencil. A drop of the material is then expressed from each tube and a small scratch, about 2 mm. in length, is made through each drop. A little practice is required to ensure uniform results, the correct depth being that which produces a faint streak of blood, showing that the skin has just been penetrated. The solutions are allowed to remain in contact with the scratch for two or three minutes and the excess is then wiped off. The results are read in twenty minutes. A positive result is indicated by the presence of a wheal, together with an area of surrounding redness. As a rule the contrast between a positive and a negative result is so clear that there is no doubt; when a result is indefinite, it should be classed as negative. In ordinary cases, when many tests are being made simultaneously, it is not necessary to use a control solution, for the contrast between the positive and negative reactions indicates the results sufficiently clearly. When only one or two tests are being made a control solution should always be employed. The swelling persists for a few hours and all traces should have disappeared in three or four days.

(d) Blood examination may reveal an eosinophilia when an allergic factor is present.

(e) An *X-ray of the chest* should always be taken to determine the presence of organic disease. Should there be any suggestion of bronchiectasis, a *lipiodol* examination may be done. The information to be gained from screening and from double-exposure films have been considered (pp 52, 69).

(f) A *fractional test meal* should be carried out as a routine. In about one-third of all cases it will be found that there is little or no secretion of hydrochloric acid.

(g) In certain cases, when a thyroid factor is suspected, the basal metabolism may be estimated.

*Treatment.*—This must be selected for the individual patient, and it depends upon the accurate assessment of the factors concerned. It may be stated that, of all the various lines of treatment at our disposal, efforts at desensitization are the least effective. It happens that, in most cases, the presence of

an allergic factor merely indicates that there is an inborn predisposition to asthma and the interests of the patient are best served by attempts to deal with the exciting factors which actually cause the attacks.

The allergic factor may be treated by either of two methods:—

(a) *Avoidance of the offending substance.*—When this is known, and when it appears to be the most important factor in producing the attacks, it may be possible for the patient entirely to avoid it. For instance, some individuals only have an attack when they come into contact with certain pollens which have a local distribution; in such, this method may be all that is required. When the cause is not so clear, an attempt may be made, by omitting certain articles of food from the diet, to arrive at the cause and so to avoid the effect. This method, although widely practised, is not very successful. It may be desirable entirely to change the environment and to send the patient abroad, preferably to a moderate altitude. Although frequently successful, this method entails a serious dislocation of the patient's life and it is therefore not usually applicable.

(b) *Active desensitization* may be specific or non-specific. The aim of specific desensitization is to raise the patient's immunity to the substance to which he is sensitive by means of a course of subcutaneous injections of minute quantities of the protein concerned. If the patient is sensitive to one substance only, especially a substance with which he frequently comes in contact, this method may be reasonably employed although the results are not really satisfactory. When respiratory infection plays a prominent part, it may be possible to benefit the patient by means of an antogenous vaccine. This is far the most satisfactory method of specific desensitization which we have at our disposal, and it is always worth adopting it when the organisms present in the sputum are suitable. The results from vaccines containing *Micrococcus catarrhalis*, *H. influenzae*, and streptococcus are good when the infection is mainly pneumococcal, the results are less satisfactory in all cases it is desirable to start with small doses and not to exceed 1/2 in. T.

In the treatment of certain

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dust extract, or magnesium hyposulphite. It must be remembered that it is impossible to assess how much benefit is due to the substance injected, and how much to the fact of the injections; the results of control experiments with blank injections suggest that the treatment depends to a large extent upon its psychological effect. The simplest material is Old Tuberculin, as suggested by van Leeuwen, and the course is given as follows:—

Weekly subcutaneous injection of  $\frac{1}{10}$  c.c. of a 1 in 1,000,000 dilution of Koch's Old Tuberculin are given for three months. The dose need not be increased. The interval between the injections is then increased to a fortnight for another three months, and then, after three months' rest, the whole course is repeated. Tuberculin is not given because it is thought that asthma necessarily has anything in common with tuberculosis; when there is evidence of active tuberculosis in the lung, it is imperative that this should be treated by such methods as seem appropriate.

(c) Non-specific therapy may be attempted in certain other ways. A simple method is a course of autohaemotherapy. Blood is withdrawn from the patient's vein and injected intramuscularly into the buttock twice weekly, starting with 1 c.c. and increasing to a maximum of 10 c.c.s. There are no reactions as a rule and the results are quite good in most allergic conditions. More vigorous methods, such as *protein shock*, may be tried in difficult cases. It is usual to inject T.A.B. vaccine intravenously, starting with 20 millions and increasing gradually to 500 millions, being guided as to successive doses by the reaction which follows each injection. Treatment by inducing pyrexia is sometimes successful; malaria, relapsing fever, or the artificially produced "electric fever" have all been employed. The decision to infect the patient with a disease is always a serious one, and care must be taken that the disease selected is one which can be readily controlled and cut short at the required time.

*Treatment of the upper respiratory factor* is all that is necessary to relieve the asthma in certain cases. If the mucous membrane is congested and sensitive, a course of hygienic treatment may be useful. The patient is advised to use twice daily a spray containing 2 per cent ephedrine and  $\frac{1}{2}$  per cent potassium sulphate, in water, to be followed after five minutes

by a simple alkaline nasal douche. *Zinc ionization* of the nose is very helpful, especially when there is recurrent catarrh, paroxysmal rhinorrhoea, or hay fever. In such cases nasal treatment frequently abolishes the asthma. When there is gross sepsis, adequate drainage should be secured, but it is more difficult to decide the proper treatment of nasal polypi, hypertrophied turbinates, and deflected septum. It is a fact that asthma frequently starts for the first time after an operation for one of these conditions and, unless there is gross obstruction, it is better to try the simpler methods before considering radical operations.

*The treatment of the broncho-pulmonary factor* is really that of the disease concerned. This is commonly bronchitis, which may be treated by advising change of environment, by inhalations, or by vaccines. When bronchiectasis is present, the outlook is not so good, although a warm dry climate, and posture, are often effective in relieving the asthma. Treatment of active tuberculosis should follow the routine for that condition; the asthmatic state is frequently improved as well. Tuberculin treatment should not be undertaken in these cases except by those who are accustomed to using this substance.

*Breathing exercises.*—It is very important to teach the patient to breathe correctly. Normally the diaphragm is the chief muscle of inspiration during quiet breathing; the intercostal muscles fix the ribs and so keep the chest wall expanded while the diaphragm descends. When inspiration is more vigorous the intercostals act more strongly and raise the ribs, thus increasing the diameters of the thorax. During forced breathing the accessory muscles of respiration are brought into play and tend to lift the upper part of the thorax away from an almost fixed diaphragm.

In the attack of asthma it is comparatively easy to draw air in through the narrowed tubes, but difficult to expel it. The lungs therefore become distended. After many attacks the muscles may become adapted to their new position even in the intervals between attacks. This condition may eventually become permanent and irremediable, but there are many patients with this deformity who can remedy it by a voluntary effort. In this group it is still possible, by means of selected *breathing exercises*, to restore the function of the respiratory muscles to a great extent.

Ordinary "deep-breathing" exercises are designed to increase the chest expansion and it will be readily understood that they cannot help the asthmatic, whose chest is already distended. The proper exercises are those which encourage mobility of the diaphragm and the lower part of the chest, and chief attention at first must therefore be fixed on efficient emptying of the lungs, especially the lower lobes. There are several suitable series of exercises. Instruction should always be given at first by a specially trained masseuse and the exercises must then be carried out by the patient over a long period. A very useful booklet on physical exercises for asthma is published on behalf of the Asthma Research Council.

*Treatment of the alimentary factor* is most important. In children it is useful to give one drachm of glucose after food. When there is no secretion of hydrochloric acid, this may be supplied in doses of 30 minims, to be taken, well diluted with water, at meal times. The diet should be light and easily digested, and the chief meal should be taken in the middle of the day. All foods which are likely to cause flatulence must be avoided, liquids must be taken between meals, and rest after meals is beneficial. Plenty of fruit may be taken, and laxatives, such as senna, may be needed to overcome the tendency to constipation. Colon lavage is often effective when the large bowel does not function well.

The endocrine factor is perhaps the most difficult of all to treat although, with the rapid advances which are being made in the preparation of extracts of endocrine glands, there is every hope that we shall soon be able to correct the endocrine factor in a proportion of cases. At present all that need be said is that where there is obvious ovarian dysfunction an attempt may be made to produce improvement by injecting one of the recently prepared extracts. When there is frank hyperthyroidism, iodine may be beneficial.

The psychological factor frequently holds the key to successful treatment and it must be approached very cautiously, for it is as easy to do harm as good. The patient's difficulties are commonly environmental, and therefore a complete change of scene or occupation may be expected to produce improvement in certain cases. When it is not possible to alter the environment, much may be done by regulation of the patient's own life, laying great stress on adequate recreation. Many patients are



## CHAPTER 17

### BRONCHIECTASIS

THE term "bronchiectasis" indicates a state of dilatation of the bronchial tubes. In the not very remote past it was used to indicate a clinical condition which was characterized by severe cough and expectoration of large quantities of foul sputum. It is now recognized that this clinical state is not necessarily present in all, or even in the majority, of cases of bronchiectasis, and the clinical picture is not nearly so clearly cut as would be imagined from the older descriptions. Bronchiectasis may be congenital or acquired.

1. The *congenital* form is the less common and always results from a maldevelopment. The embryonic lungs are largely composed of interstitial tissue traversed by bronchial tubes, the ends of which form buds. The act of breathing results in the formation of large infantile alveoli in comparatively small numbers. During the first few years of life new tubes and alveoli continue to develop in the peripheral part of the lung, and full development is not reached until the ages of 6 and 12 years.

(a) The large infantile alveoli may persist and, by dilating, may form *cysts* (figs. 15, 17). These cysts may be single or multiple. They are usually confined to one lobe, but there is a very rare condition in which they are diffusely scattered throughout both lungs ("honeycomb" lung).

(b) Alveoli may fail to develop from the growing ends of the bronchioles which dilate and form cylindrical tubes.

Both of these types may sometimes be present together. Congenital bronchiectasis may occur in any part of the lung and the cavities may be single or multiple. Frequently they are dry and air-containing. Rarely they are filled with fluid. The pleura is usually free from adhesions, and this fact is of practical importance in the surgical treatment of bronchiectasis. Other congenital abnormalities may be found in the same patient.

2. *Acquired bronchiectasis* is the commoner form. It is by no means certain how the condition develops but various factors appear to enter into the pathogenesis.

*Weakening of the walls of the bronchi* is generally agreed to be a most important factor and infection is the usual underlying cause of this. Bronchiectasis is a common sequel of measles, whooping-cough, influenza, and bronchopneumonia; chronic tuberculosis may produce local dilatation. The common association of sinusitis with bronchiectasis must be kept in mind, not only because the upper respiratory infection plays a part in causing the disease, but also because careful treatment of the sinus condition is often necessary before carrying out an operation for removal of the diseased part of the lung. There is no specific bacteriology of bronchiectasis and it would appear that any of the organisms which commonly invade the bronchi are capable of weakening their walls.

*Pressure changes.*—The bronchi in a collapsed lobe are often found to be dilated. This is difficult to understand and the cause is disputed. When the condition is congenital (atelectasis) it would appear that the bronchi are normal in size at birth and that any subsequent dilatation is due to infection. When the collapse is acquired, as in cases of bronchial tumour, foreign body, or external pressure on the bronchus, the factor of infection appears always to be present.

In some of the more acute cases it is stated that a plug of mucus is sucked into the peripheral bronchioles, blocking them and causing patchy collapse. The larger bronchi are patent and are subjected to atmospheric pressure, and the negative pressure in the thorax is increased owing to the collapse. Suction is thus directly transmitted to the bronchi through the inelastic collapsed lung and they dilate.

Both the inspiratory and expiratory phases of the act of coughing have been considered to be possible factors in causing bronchial dilatation but there is little evidence that either plays more than a very minor role.

Fibrosis of the lung is often present but it is the result of the inflammatory condition which caused the bronchiectasis as a rule. Of course fibrosis may sometimes cause bronchial obstruction and so increase the bronchial damage. There is nothing to suggest that contracting fibrous tissue ever pulls apart the bronchial walls.

In the early stages the disease may be limited to comparatively few bronchi, although spread of the infection and retention of secretion may cause extension of the disease in

the course of time. The earlier the condition is seen, the more likely it is to be confined to a single lobe. In the acquired type the lower lobes are usually affected, but in the congenital type, and in cases of pneumoconiosis, any part of the lungs may be involved.

There are two main anatomical varieties : the *saccular* and the *cylindrical*.

In the saccular type there is great variation in the size of the cavities, which may be from half an inch to 3 or 4 inches in diameter. They vary greatly in number, from a single cavity upwards. The condition is most commonly found in the terminal bronchi, and is usually congenital in origin.

Cylindrical dilatation tends to be uniform, and the smaller divisions of the bronchi retain the size of the parent tubes. In some cases the bronchi increase in size towards the periphery, giving rise to an appearance resembling that of the fingers of a glove.

Changes in the lungs are common and very diverse. There may be much emphysema and fibrosis, and there is often pulmonary suppuration or a terminal bronchopneumonia. In some cases, especially in the congenital form, the surrounding lung tissue may be normal. In addition to being a causal factor in some cases tubercle may occur as a complication of bronchiectasis, and in such cases the tuberculous lesions are not very prominent. The rupture of a subpleural congenital cyst is a common cause of *spontaneous pneumothorax*.

Absorption of septic material leads to *clubbing of the fingers* and sometimes of the toes. Much less commonly there is swelling of the wrist and ankle joints as well as some of the larger joints, a condition known as *hypertrophic pulmonary osteo-arthritis*.

The pulmonary circulation is not affected in the milder types but, when the disease is advanced, and especially when there is much fibrosis, the right side of the heart may dilate. Clinically, it is important to be sure whether shift of the right border of the heart is due to dilatation or to displacement.

In chronic suppurative cases the absorption of toxins may lead to *amyloid disease*. The liver and spleen may be found to be enlarged, firm, and smooth, and there may be massive albuminuria. The skin may assume a typical waxy appearance and there may be chronic diarrhoea.

## BRONCHIECTASIS

133

A common and fatal complication is *brain abscess*. The infection is embolic, but it is not known why the brain should be so frequently picked out.

The clinical picture varies very much and the cases may be divided into four groups: symptomless, bronchitic, haemorrhagic, and suppurative.

1. *There may be no symptoms of any sort and the condition is discovered accidentally during a routine physical examination, or at post-mortem in patients who have died from some other condition.* Symptoms are liable to occur at any time, especially as a consequence of a descending infection of the respiratory tract.

2. The picture may be that of *recurrent bronchitis*. The recurrent infection may be either acute or chronic, and symptoms are more common in the winter. The clinical diagnosis in these cases is frequently "bronchitis", and the real nature of the condition is usually not suspected, for there may be few or no physical signs. A routine X-ray of the chest in such cases may reveal the true state of affairs, but a lipiodol examination will be necessary in order to clinch the diagnosis. The presence of clubbing of the fingers in cases of chronic bronchitis is always suggestive of developing bronchiectasis. It must be remembered that two separate conditions are included in this clinical group: (a) cases of congenital or of old standing acquired bronchiectasis, which have the symptoms of recurrent bronchial infection, and (b) cases of chronic bronchitis which are developing bronchiectasis; this group is more likely than the former to be bilateral.

3. The *dry haemorrhagic form* is fairly common and frequently causes errors in diagnosis. There are recurrent haemoptyses, and the patient may cough up from several ounces to a pint of blood at intervals which vary from several weeks to several years. Between the attacks the patient may appear to be in perfect health, or there may be symptoms of recurrent bronchitis. In some cases the condition may be confused with tuberculosis, and repeatedly negative sputum examinations in cases of persistent haemoptysis should always suggest the possibility of this type of bronchiectasis. The physical signs caused by the dilated bronchi are often not conspicuous and the diagnosis must be made by the bronchogram. The history of some antecedent infection, such as bronchopneumonia in childhood, is suggestive.

4. The clinical picture of the *suppurative type* which, up to the time of the introduction of lipiodol in diagnosis, was regarded as typical bronchiectasis (figs. 19, 20), is one in which the patient coughs up from several ounces to a pint of foul sputum daily. In reality this symptom is characteristic of one comparatively rare type of bronchiectasis. Gross infection of a bronchiectatic area may lead to suppuration, with the production of a large quantity of pus. If the infecting organisms belong to the pyogenic group the sputum will be purulent but there will be little odour. Foul sputum always indicates an anaerobic infection, usually with organisms of the fusospirochaetal group. This infection may occur early or late in the course of the disease and it is always of serious significance. The symptoms consist of severe cough with production of large quantities of the typical sputum, occasionally haemoptysis, progressive shortness of breath, and frequently loss of weight. The cough is often brought on by change of position. The appearance of the patient is suggestive of toxæmia; the skin is sallow and moist, there is clubbing of the fingers, and often an unpleasant odour of the breath.

On physical examination the signs in the chest are very varied. With unilateral disease the signs may be those of fibrosis. There is wasting and limitation of movement on the affected side, the heart and trachea are displaced towards the side of the lesion, the percussion note is often impaired on account of the fibrosis. The breath sounds vary tremendously. There may be bronchial breathing, bronchophony, and coarse rales. Should the cavities be full of pus at the time of the examination there may be absent breath sounds and no added sounds. A suggestive feature on auscultation is that the coughing up of sputum may alter the physical signs appreciably. In fact the physical signs vary so much that, although positive signs may be accepted with confidence, the absence of physical signs does not necessarily exclude the possibility of bronchiectasis.

An acute form of bronchiectasis may develop in a collapsed lobe following major operations or the inhalation of a foreign body. The clinical picture in the early stages may resemble that of massive collapse or lung abscess and a bronchogram will be necessary in order to make an exact diagnosis.

*Special investigations.*—In advanced cases the clinical diagnosis of bronchiectasis is usually fairly easy. In early cases,

and in those in which the symptoms and signs are anomalous, special investigations must be employed.

1. *The sputum.*—In the bronchitic type an ounce or two of mucopurulent sputum is expectorated daily. Examination shows nothing more than is found in cases of bronchitis. The copious sputum characteristic of the suppurative type is said to settle into three layers on standing, an upper layer of froth, clear liquid in the middle, and granular debris at the bottom. This type is not often seen and usually the sputum looks like, and behaves like, pus. Microscopically the chief constituents are pus cells, epithelial cells, and mucus. In some cases small fibrinopurulent casts of the bronchioles, Dittrich's plugs, may be seen. Elastic tissue should not occur in the sputum in bronchiectasis.

The bacteriology is very diverse. Tubercle bacilli must always be sought, for tuberculosis is a differential diagnosis of bronchiectasis, and the two conditions may coexist. Most of the organisms usually found in the respiratory tract may be present in the sputum, but the only ones which are at all characteristic are fusiform bacilli, spirilla, and anaerobic bacilli, many of which are likely to be penicillin sensitive.

2. *The X-ray appearances* are equally varied. Thin-walled cavities may be clearly seen, or there may be simply an area of apparent fibrosis. Sometimes there is a diffuse haziness suggestive of early tuberculous infiltration. When the disease is situated in the left lower lobe, there may be a triangular shadow behind the heart, which indicates collapse of the left lower lobe (fig. 21), or there may be very little to be seen. *An appearance of increased translucency above the left diaphragm, which indicates localized emphysema in this region, is very suggestive of fibrosis or collapse, and possibly bronchiectasis, of the left lower lobe.*

3. The only certain method of determining the extent of bronchiectasis is by a lipiodol examination. In order that this may be successfully carried out, it is necessary that the patient should always be postured before the oil is given, so that the cavities are as empty as possible. The X-ray may show cystic spaces partly filled with oil (figs. 16, 17), or there may be fusiform or "glove" dilatations (figs. 19, 20). Lipiodol should enter the diseased area without difficulty. In cases of lung abscess, which may present a similar clinical and radiological picture, the oil usually does not enter the cavity (fig. 18). Lipiodol is given in order to determine the extent of the damage, and it must be remembered that the lateral view is just as important as the antero-posterior for this purpose; each side must therefore be examined separately. In cases where surgery is being considered it is as important to outline the apparently sound bronchi as it is to investigate the diseased area. Operation should rarely be considered when there is evidence of damage in both lungs.

4. A *bronchoscopy* is not of very much value in diagnosis, but it will reveal an unsuspected foreign body, a carcinoma, or a bronchial stricture, in a certain proportion of cases. It is therefore an investigation which is frequently desirable. In simple cases the mucous membrane of the bronchi leading to the affected part is reddened and swollen, and pus may be seen exuding from this area.

The *treatment* of bronchiectasis, even though it be confined to a single lobe, when there are no symptoms except perhaps an occasional attack of bronchitis, should be directed to improving the general health of the patient and to increasing the resistance to infection. An autogenous vaccine during the winter months may be useful. Severe operations, such as lobectomy, are hardly ever justifiable in this type. Cases are reported in which a slight degree of bronchiectasis has disappeared spontaneously.

The treatment of cases in which recurrent haemoptysis is the chief symptom is a matter of some difficulty. If the haemorrhages are not very severe, with an interval of months, or even years, between them, the patient will doubtless refuse to have radical treatment and will elect to suffer the mild inconvenience of being confined to bed for a few days at intervals. Treatment of the haemoptysis consists chiefly of rest in bed. Severe recurrent haemorrhage is an indication for lobectomy or for such other surgical procedure as may be thought necessary.

In the suppurative group the effect of *postural drainage* should first be studied. In bilateral cases this is the only method of treatment which offers any prospect of improvement. The posture advised will depend entirely upon the site of the damage. Special "posture beds" are available and the posture required may be maintained for increasing intervals, up to 18 hours daily. When efficient drainage is being obtained it may be necessary for the patient to sleep on the posture bed for an indefinite period, leading a normal life by day.

If cultures show that the majority of the organisms present in the sputum are penicillin sensitive an attempt may be made to reduce the intensity of the infection by means of penicillin inhalations. This may be done by using an atomizer which contains 20,000 units of penicillin dissolved in 1 c.cm. of normal saline, and the dose is repeated three or four times in the day. Alternatively, the same amount of penicillin may

be emulsified in 2 or 3 c.cms. of olive oil and introduced in the manner described for the oral route for lipiodol (p. 65). Although the immediate results are often quite good, there is a tendency for the infection to recur as soon as the treatment is discontinued. Sometimes the primary organisms disappear, only to be replaced by penicillin insensitive coliform bacilli.

Vaccines are sometimes useful and the intratracheal injection of a 1 per cent solution of oil of gomenol in olive oil has the effect of diminishing the sputum and rendering it less offensive. Bronchoscopic lavage has been advised in order to keep the cavities as empty as possible. This method of treatment is suitable only for bilateral cases, and it is doubtful if the results are in any way superior to those obtained by postural drainage. In all of these cases an open-air life in the country is obviously preferable. As suppuration in the nasal sinuses is frequently responsible for keeping up the active bronchial infection, it is essential that any sinusitis present should be dealt with as effectively as possible, and before surgery is seriously considered.

Great advances have been made in the surgical treatment of bronchiectasis in the last few years. When there is definite disease in a single lobe, with more than an ounce of sputum daily, *lobectomy* is the treatment of choice. More than one lobe may be removed, and it is even possible to remove the whole of an affected lung (pneumonectomy). It is in this connection that the significance of the anatomical distribution of the bronchi into segments (p. 53) is becoming increasingly appreciated. In quite a number of cases bronchography may reveal dilatation of bronchi in both lungs, but the total extent of bronchiectasis is not necessarily great. It is possible, by segmental resection of the affected parts, to remove substantial areas of disease from both lungs.



## CHAPTER 18

### TUMOURS OF THE BRONCHI AND LUNGS

#### Bronchial Carcinoma

By far the commonest primary intrathoracic growth is *bronchial carcinoma*. Recent statistics leave no doubt that this variety of tumour is much commoner than it was thirty years ago, and there is evidence to show that, in some districts at least, the incidence is still increasing. The reason for this spectacular change in frequency of bronchial carcinoma is still a mystery. It is slightly comforting to reflect that the possibilities of thoracic surgery have been greatly extended in the last few years, and that there is now the prospect of successful removal of a malignant lung tumour, always provided that the growth is recognized before vital structures are involved and before metastasis has taken place.

Very little is known of the cause of this type of cancer. Chest injury does not appear to be a factor. Inflammatory diseases, such as pneumonia and influenza, have been suspected, but convincing evidence is lacking. Chronic infections, such as tuberculosis and syphilis, do not appear to be in any way concerned, and there is little evidence to suggest that irritation from tobacco smoke, tar products, or silica play any part in causation. There is only one chronic irritation which is definitely known to cause bronchial carcinoma, and that is the inhalation of radio-active dust from the cobalt mines of Schneeberg in Saxony, where the disease is common amongst the miners.

The condition is four times as common in men as in women, and it occurs chiefly between the ages of 40 and 55, although it may occur before the age of 20 and in extreme old age.

*Pathology.*—Growths arise from the mucous membrane of the bronchus, and it must be remembered that this is a stratified membrane composed of two or three layers of cells (fig. 63). The superficial ciliated columnar cells rest upon deeper rounded and oval cells, thus forming a layer of *transitional epithelium*. The bronchial tree is derived from the primitive hypopharynx, which is lined by stratified squamous epithelium. During development of the respiratory tract the character of the

epithelium alters from squamous to columnar and, when this is kept in mind, it is easy to understand the histology of bronchial carcinoma. The usual histological types are :—

1. "*Oat-celled*" carcinoma.—This is the commonest type. The tumour arises in the deeper layers of the bronchial mucous membrane. The cells are oval or round and the general appearance is similar to that of a sarcoma or an endothelioma (fig. 66). Indeed, until a few years ago, there was much discussion as to the nature of these tumours but it is now recognized that they are bronchial in origin. They are, in fact, comparable to the basal-celled carcinoma of the skin and to some of the malignant tumours which arise in the urinary tract.

2. *Columnar-celled carcinoma*.—As would be expected, this is fairly common (fig. 64).

3. *Squamous-celled carcinoma*.—This is less common, and results from a process of metaplasia (fig. 65). There is no substance in the suggestion that these tumours arise from squamous-celled "rests" which have been postulated in the mucous membrane of the bronchi.

4. *Spheroidal-celled carcinoma*.—This rare type of tumour arises in the secreting glands which are situated beneath the basement membrane.

It is desirable to bear these histological appearances in mind in view of the ever-increasing use of the bronchoscope in obtaining material for examination.

The majority of the tumours arise in the main bronchi, and the two lungs are equally affected. The lower bronchi are involved rather more commonly than the upper. Four distinct anatomical varieties are found :—

Type 1. *The hilum tumour*.—This type takes origin in the main bronchus and remains limited to this region, forming a mass in the hilum of the lung (fig. 27). Sometimes the tumour projects into the lumen of the bronchus and causes complete obstruction, with consequent collapse of the lung (figs. 13, 22).

Type 2. *The lung tumour*.—In this type the growth takes origin from a bronchiole deep in the substance of the lung (figs. 28, 30). It usually forms a rounded mass and, provided that the diagnosis can be made sufficiently early, it may be amenable to surgical removal. Tumours of this type sometimes break down and discharge their contents into a bronchus ("malignant abscess"). The X-ray shows a fluid level in the mass and care must be taken to distinguish this from a simple lung abscess.

**Type 3. The mediastinal tumour.**—A growth which takes origin in one of the larger bronchi may spread towards the mediastinum instead of infiltrating outwards into the lung (fig. 29). It is difficult to recognize this type at first sight because it so closely simulates a primary mediastinal tumour, which is not a carcinoma but a sarcoma or endothelioma. The latter types are much more radio-sensitive than carcinomata, and the result of a course of X-ray treatment may be taken into account in attempting to establish an exact diagnosis in difficult cases (compare figs. 29, 57, 58).

**Type 4. The combined tumour.**—Sometimes a growth which commences in the region of one of the larger bronchi may infiltrate in both directions simultaneously, forming a mass extending from the lung to the mediastinum.

These types are based on an analysis of post-mortem material and they are clinically distinct. The hilum tumour tends to remain localized and death often results from metastases, whereas the mediastinal tumour tends to cause death by compression of vital structures. Infiltration may occur either by direct spread of the tumour towards the lung or mediastinum, or by way of the lymphatics, in which case the growth is seen to protrude outwards into the lung tissue in a fan shape, and section of the bronchi shows the appearance of tubes sheathed in a mass of white tissue.

The presence of a growth in the lumen of a bronchus may give rise to three main complications: *collapse of the lung*, *suppuration*, and *haemorrhage*.

The pleura is commonly involved, either by direct spread of the growth to the visceral part of the membrane, or as the result of obstruction and infiltration of the lymphatics. The chief changes which are found in the pleural cavity are:—

- (a) Dense adhesions, which may contain malignant cells.
- (b) Malignant infiltration and pleurisy.
- (c) Clear effusion.
- (d) Empyema.
- (e) Chylothorax is very rare but it may occur on the left side if growth involves the thoracic duct.

Metastases occur in the majority of cases and the common parts involved are:—

1. *Lymphatic glands.*
  - (a) In the hilum of the lung.
  - (b) Above the clavicle.
  - (c) In the axilla.

- (d) In the upper abdomen. It is very often from these latter glands that the abdominal spread takes place.
2. *In the abdomen.*—The organs most commonly affected are the liver, suprarenals, pancreas, kidneys, and peritoneum.
3. *In the nervous system*—Deposits are commonly found in the brain and they are often multiple. The spinal cord may be involved, either by direct extension of the growth through an intervertebral foramen, or as the result of metastasis.
4. *Deposits in bone* are fairly common, the long bones, pelvis, ribs, sternum, and scapulae being those chiefly affected.
5. *Nodules* may be found in the skin, especially of the trunk and of the scalp.

Sometimes metastasis occurs so early as to cause symptoms before there is any evidence of the primary growth in the bronchus. In such cases the diagnosis of malignant disease may be correctly made, but the metastatic nature of the tumour may not be recognized. This applies especially in cases of intracranial tumour, and it should be an invariable rule to take an X-ray of the chest before embarking on operations for cerebral tumour.

Bronchial carcinoma may present itself in a variety of ways. The symptoms may date from an acute infection labelled "influenza" or "pneumonia". Sometimes the first symptoms are those of acute pleurisy, usually with effusion. In most cases, however, the symptoms appear so gradually that it may be months before it is realized that there is anything seriously wrong in the chest.

*Cough* is a common early symptom. It may have no distinguishing character from that of bronchitis, or it may be paroxysmal, suggesting bronchial irritation. A "brassy" cough should always suggest the presence of a mass pressing on the trachea. Finally, the cough may become husky after a time. This occurs when the recurrent laryngeal nerve is involved either by growth or by an aneurysm.

The *sputum* is not copious as a rule. Should the lung tissue distal to the growth break down, there may be a considerable quantity of offensive sputum. *Haemoptysis* is extremely common and the sputum is usually pink at some stage. In some cases the sputum is said to resemble "red-currant jelly", an appearance which is very rare and of little diagnostic importance.

*Pain* is a common symptom when the growth is situated

near the pleura. It may result from the strain of repeated coughing, from infiltration of the pleura, from invasion of the intercostal nerves, or from pressure on a posterior root ganglion when the spinal cord is involved. When deposits occur in the ribs there may be severe local pain.

*Dyspnoea* occurs in most cases sooner or later, but it is not a common early symptom. It depends to some extent on how rapidly the lung tissue is thrown out of action. Rapid accumulation of fluid in the pleura, or sudden collapse of a lung, may cause urgent dyspnoea, whereas the same conditions occurring more slowly may cause little distress.

In the later stages an element of heart-failure may enter into the causation of dyspnoea.

The *weight* is often surprisingly normal for a considerable time, but patients vary very much in this respect.

Symptoms resulting from intrathoracic pressure are not uncommon. *Alteration in the voice* is frequent from infiltration of the recurrent laryngeal nerves. Stridor is diagnostic of tracheal compression; *dysphagia* may occur, and there may be symptoms of obstruction of the great veins, such as *oedema* of the arms and face.

The *physical signs in the chest* present themselves in four main groups:—

1. *Signs of collapse*.—A single lobe or a whole lung may be involved. The signs are naturally very extensive, but it does not follow that the tumour is necessarily large, as a comparatively small growth may completely occlude a bronchus. The affected side will be seen to be somewhat retracted and to be moving badly or not at all. The heart and trachea are displaced *towards* the affected side, the percussion note is grossly impaired, and the breath sounds are absent. The signs simply indicate collapse of the lung, but, in a middle-aged patient, growth is by far the commonest cause of this condition.

2. *Signs of consolidation*.—The affected side may not be moving well and there may be some retraction. The heart and trachea are not displaced, and the percussion note varies, but is usually considerably impaired. The breath sounds differ according to the state of the lung. With dense consolidation and bronchial obstruction they may be weak or absent; in consolidation of a bronchus there may be bronchial breathing. If the lung is consolidated and formed an abscess cavity, there may be bronchial breathing and rales. The correct diagnosis should be reached by consideration of the symptoms and the presence of metastases.

## TUMOURS OF THE BRONCHI AND LUNGS 143

3. *Signs of mediastinal obstruction.*—In these cases there may be retrosternal dullness, together with signs of collapse or consolidation of a part of the lung. The most important signs, however, are those of pressure upon vital structures. There may be oedema of the face or of either arm, there is often dilatation of the veins of the chest or abdomen on the side of the obstruction, the pulses may be unequal, and there may be paralysis of a vocal cord. Apical tumours may cause the cervical sympathetic and the pupil dilated, whereas later, in the early stages, when there is sympathetic irritation, the eyeball on the affected side is protruded and the pupil dilated, whereas later, when the nerve is paralysed and the pupil dilated, there is never contracted pupil. Most of these signs may also result from aneurysm or from any other type of mediastinal tumour. There is never tracheal tugging nor asynchrony of the pulses with growth, but exact diagnosis is frequently difficult. The patient should always be observed on the X-ray screen, for pulsation in the swelling is strong evidence of aneurysm, although absence of pulsation does not entirely exclude it. A lymphogram will help to distinguish between expansile and transmitted pulsation. Paralysis of the diaphragm is highly suggestive of malignant growth.

4. The patient may present the physical signs of pleural effusion. Any middle-aged patient who has fluid in one pleural cavity for no apparent reason is likely to have an underlying growth. The chest will not be retracted, but movement will be limited; the heart and trachea will be displaced away from the affected side, vocal vibrations will be absent, the percussion note will be dull, and breath sounds will be inaudible. Exact diagnosis involves the consideration of the special investigations.

The final diagnosis must always be dependent upon microscopic examination of tissue, but other investigations may also be extremely helpful.

1. *The sputum.*—*Tubercle bacilli* are found in about 10 per cent of cases of malignant growth. A positive result does not therefore exclude the possibility of carcinoma. Elastic fibres are frequently seen when there is a destruction of lung tissue. Malignant cells have been recognized by some observers, and this is the simplest observation which can provide a definite diagnosis, but the recognition of malignant cells is not yet sufficiently certain to allow this method to be used as a routine.

2. *X-rays.*—The X-ray appearance varies according to the site of the growth and the consequent effect upon surrounding structures. In some cases there is a typical picture of collapse of a lobe (fig. 22), or of a lung (fig. 13). In others there is an irregular rounded shadow in the lung field. This may be homogeneous (fig. 28), or there may be the appearance of a fluid level, simulating lung abscess. When a tumour is situated behind the heart it may be impossible to define

unless a lateral view is taken. There may be a mass in the mediastinum (fig. 29), or pleural effusion. None of these appearances are conclusive, but a paralysed diaphragm, in addition to the other findings, is highly suggestive of growth (fig. 30); the fact of phrenic paralysis must be confirmed by screening. The main conditions which have to be differentiated from growth are *aneurysm*, *abscess*, *tubercle*, *bronchiectasis*, *gumma*, and *secondary growth*, and investigations which are appropriate to each condition must be carefully carried out, always bearing in mind that a growth may be present in addition to one or more of these diseases.

The presence of a large quantity of fluid interferes with the radiographic investigation of the lung condition, and it is sometimes necessary to remove the fluid in order to repeat the X-ray. When there is much fluid an air replacement may be done.

A *tomogram* will sometimes throw light on an obscure case.

A *bronchogram* is rarely of great diagnostic value, for the reason that, if the growth is advanced, and in a main bronchus, the diagnosis can be more surely reached by other means, whereas, when the growth is situated towards the periphery of the lung, the bronchogram is not helpful. Complete obstruction of a large bronchus by a growth gives the appearance of a cone-shaped deformity; the block may be best seen in a lateral view (fig. 32). The real value of lipiodol is, firstly, that it may show that the suspected bronchus is completely patent, and, secondly, that in the rare cases in which a very early carcinoma is present the examination may demonstrate a block in a secondary bronchus which is out of reach of the bronchoscope. It is in this latter group that lipiodol may be of the utmost value, for it will demonstrate the presence and site of early bronchial obstruction in time for radical removal to be attempted with some hope of success. Lipiodol does not as a rule enter a lung abscess cavity and it is therefore of little value in distinguishing between abscess and growth.

3. *Bronchoscopy*.—This is by far the most certain means of establishing the diagnosis, for a growth may actually be seen and a piece may be removed for microscopic examination. Unfortunately, growths which are visible through a bronchoscope are so near the hilum that they are usually inoperable. A routine bronchoscopy should, however, be undertaken in all cases in which the presence of a growth is suspected.

4. *Biopsy*.—Tissue may be removed through a bronchoscope, or material for examination may be obtained by removing a superficial gland or a nodule from the skin. A positive result is conclusive.

5. *Blood examinations*.—The blood count is of little assistance. Some patients are anaemic, and the *leucocyte count* is often raised. The sedimentation rate is usually high, and the Wassermann reaction is sometimes positive. Pulmonary syphilis is a rare condi-

tion and a positive Wassermann reaction is not conclusive, but the result must be taken into consideration when aneurysm is suspected.

6. *Examination of pleural fluid.*—Pleural effusion is fairly common at some stage, and it may be the earliest sign of disease. The fluid may be either straw-coloured or blood-stained; rarely there is pus. Under the microscope Foulis cells may be seen. These are multinucleate and have a deeply staining cytoplasm; they are not malignant cells but are thought to be altered endothelial cells which have been shed into the fluid.

7. *Thoracoscopy.*—After a pneumothorax has been induced, or after the replacement of fluid by air, it is possible by means of a thoracoscope to inspect the state of the pleura. Nodules of growth may be seen and it may even be possible to remove material for section. This method is not often used.

8. *Exploratory thoracotomy.*—In cases where the suspected tumour is known not to be situated near vital structures it is sometimes desirable to open the chest in order to make an early diagnosis, and occasionally exploration is useful in cases in which the diagnosis lies between bronchial carcinoma and chronic lung abscess, if an abscess should be found, the exploration can be turned into a radical operation.

The average duration of life from the time of appearance of the first symptom is a little less than one year, and treatment is almost hopeless. The onset is so insidious that the growth has usually progressed far beyond the possibilities of surgery before the diagnosis is even suspected. If the diagnosis can be made at a sufficiently early stage, always assuming that metastases cannot be found, radical removal of the tumour should be attempted if the general condition of the patient permits.

It cannot be too strongly emphasized that routine chest X-rays and mass radiography are proving invaluable in the recognition of bronchial carcinoma, and it is therefore now possible to take a somewhat less gloomy view of the outlook in the less advanced case. The operation of total pneumonectomy is now comparatively safe and it has been successfully performed on many occasions. It should therefore be considered to be the treatment of choice whenever the growth is reasonably localized, when there is no reason to suspect involvement of the vital structures in the mediastinum, and when a careful search has failed to reveal the presence of metastases. In the great majority of cases the question of surgery does not arise, and at present there is little alternative to



radiotherapy. This may be attempted either by X-rays or by radium but the results are extremely depressing. When the growth is situated in a main bronchus radium may be introduced through a bronchoscope either in a needle or, in the form of radon seeds, directly into the growth. There is some risk of perforation of a large blood vessel, but some of these growths shrink to a marked extent as a result of local radiation. The method is of little practical value. Radium needles may be inserted into a superficial growth through the chest wall but the results on the whole are poor. Better results might be expected from treatment with deep X-rays when the treatment is carried out with a good apparatus and directed by a skilled radiotherapist, but there is no evidence that radiotherapy has any appreciable effect in prolonging life on the average. The great value of deep X-ray treatment is that it can cause sufficient shrinkage of the growth to relieve the more distressing symptoms, such as irritating cough, pain, haemoptysis, dyspnoea, and oedema. This is the most useful result which can as yet be placed to the credit of radiotherapy. Occasionally a patient may live for as long as five years after beginning treatment, but such results are exceptional. This fact, however, might be held to justify routine radiotherapy, for any case may prove to be one which will respond well; we have no means at present of gauging the probable response to treatment. At the same time it must be recognized that X-ray treatment has a very debilitating effect on many patients, and it might act as the final straw in some. Particularly are X-rays to be avoided when there is any evidence of local infection, for the lung tissue is likely to break down and form an abscess.

In all cases the presence of metastases contra-indicates active treatment. In this type of cancer it would seem that what is needed is some method of general treatment, such as the injection of some chemical substance into the blood stream, in order to affect the whole of the body. Some substances have been employed in this way but they have not so far proved effective.

In all too many cases the only possible treatment is symptomatic. The usual drugs must be used in ever increasing doses. The most effective is heroin and, when it is decided that an anodyne is necessary, it should be given in adequate amount.

*Superior Pulmonary Sulcus Tumour*

This rare tumour, described by Pancoast, is included here because it closely simulates an apical lung lesion and has therefore to be distinguished from apical bronchial carcinoma, tuberculosis, and mediastinal tumour. The growth is malignant and runs a rapid course. Although little has been written about its histology it appears certain that it is a carcinoma, and Pancoast has suggested that it arises from epithelial remains of the fifth branchial cleft.

The clinical picture is very characteristic. The symptoms are those of pressure on nerves, severe pain in the shoulder, axilla, or arm, with wasting of the muscles of the hand, *Horner's syndrome* (p. 143), and voice change due to paralysis of the recurrent laryngeal nerve. On examination there may not be any physical signs and the chest X-ray shows nothing abnormal in the early stages, although later a shadow may be visible internal to the apex of the lung and there may be erosion of ribs. The phrenic nerve is sometimes involved. There is no effective treatment.

*Innocent Bronchial Tumours*

Bronchoscopy has brought to light a considerable number of innocent tumours of the trachea and bronchi. Chondroma, fibroma, papilloma, and myoma of the trachea are rare and they do not cause symptoms until their increasing size results in stridor. They may be removed through the bronchoscope.

The commonest benign bronchial tumour is an adenoma, sometimes fibroma, myoma, or angioma may be found. Adenomata bear a strong resemblance to salivary gland tumours. Many of them, however, are more or less undifferentiated and considerable experience is necessary to distinguish them from carcinomata. Local infiltration is sometimes seen but metastases do not occur. Occasionally these tumours project outside the bronchus but most are intrabronchial. Symptoms may have been present for many years. The most important is recurrent hæmoptysis, although chronic cough is also common. Bronchial obstruction and pulmonary collapse are likely to supervene and infection may occur distal to the tumour. In fact, pulmonary suppuration is the usual cause of death.

A bronchogram will show the site of the tumour, which will probably be seen to project convexly into the lumen of the bronchus (fig. 31), in distinction to the conical deformity of a carcinoma. But the only certain method of diagnosis is bronchoscopy and histological examination of a piece of tissue. Most of these tumours are pedunculated and the treatment is bronchoscopic removal combined with local irradiation. Tumours which are extrabronchial or which

cannot be completely removed, those which recur, and those which are associated with distal infection should be dealt with by lobectomy.

### *Tumours in the Lungs*

*Innocent tumours.*—Benign tumours are rare and are likely to be found on taking a routine X-ray of the chest. A true chondroma may originate in a remnant of cartilage and it may become ossified. These tumours sometimes become sarcomatous. Fibroma and leiomyoma are described.

*Primary malignant tumours.*—The existence of such tumours is still doubtful. Most of the cases described as primary sarcoma or carcinoma of the lung are in reality bronchial carcinomata. There is, however, a very small number of mucin-secreting tumours which are probably true carcinomata arising from the lining of the alveoli. These tumours appear to have a comparatively low-grade malignancy.

*Metastases in the lungs.*—Secondary deposits are frequently found in the lungs. The most common sources of the deposits are the breast, prostate, kidney, thyroid gland, stomach, and the long bones. The symptoms are haemoptysis, cough, and pain in the chest. Dry pleurisy, which is often painless, is frequently noted, as is pleural effusion. If multiple deposits are seen in both lungs on X-ray the diagnosis is obvious (figs. 33, 34). Sometimes a solitary deposit is found and it may then be difficult to distinguish from a bronchial carcinoma, or even from an innocent tumour, especially as it is often sharply defined. It is necessary to be quite sure that there is no primary growth elsewhere, especially in a kidney, before undertaking lobectomy for what appears to be a primary intrathoracic tumour.

## CHAPTER 19

### CHEST INJURIES

THE chest wall is a relatively resilient structure and the lungs are therefore well shielded from casual damage. In treatment of chest injuries the essential distinction depends upon whether the chest, that is the pleural cavity, is "closed" or "open".

*Closed injuries* are the result of external violence, crushing injuries, or blast. There may be fracture of ribs, perhaps with penetration of the lung, pneumothorax, haemothorax, or bleeding into the lung tissue. Severe injuries are always attended by a considerable degree of shock. The local symptom of injury to the lung is of course haemoptysis. Surgical emphysema is often present when there is a pneumothorax, but rarely otherwise. It may sometimes happen that injury to the lung is caused during exploration of the chest with a needle. Pneumothorax is a not uncommon result; injury to a large blood vessel is rare but, when it does occur, the resultant haemorrhage may prove fatal. Spontaneous fracture of a rib is a rare result of a severe bout of cough or of an attack of asthma.

*Open wounds* are obvious on inspection. The most dangerous type is the "sucking wound", from which air gains free access to the pleural cavity. The danger arises from the fact that the mediastinum is usually mobile and a "pendulum swing" or even "mediastinal flutter" is likely to result. In addition there is dislocation of the heart and great veins with inefficient filling of the auricles and a fall in the cardiac output.

There are many complications of chest wounds :—

#### 1. *In the pleura*

*Closed :*

Foreign body.

Pneumothorax.

Haemothorax.

Empyema (later).

*Open :*

"Sucking wound."

2. *In the lung.*

Foreign body.

Collapse of lung on either side.

Infection. Broncho-pneumonia, abscess.

3. *Shock.*

The correct treatment of chest injuries is a matter of great importance and the following principles should be followed.

The diet should be adapted to the needs of the patient. Starvation is not necessary simply because there is hæmoptysis.

Shock should be treated by warmth, and perhaps by transfusion of blood or of plasma. The continuous administration of 90 per cent oxygen by means of a B.L.B. mask is also recommended, even in the absence of cyanosis.

It is now the practice to give a course of prophylactic penicillin in order either to avert or greatly to reduce the gravity of secondary infection.

The most urgent indication for surgical treatment is the "sucking wound", which must be closed at once, if need be by a pad and strapping. It is agreed that chest wounds should be excised as soon as possible and many of them can then be sutured, although the length of time which has elapsed from the receipt of the injury must be taken into account. Foreign bodies should always be located by X-rays and most of them should be removed if accessible, and if the condition of the patient permits. The treatment of hæmothorax and pneumothorax are considered in a later section.

The value of breathing exercises in promoting subsequent expansion of the collapsed lung is not universally accepted. It is certain that the common "blowing" exercises are useless, but it is possible that graduated physical exercises designed to promote mobility of the affected side of the chest may assist somewhat in causing the lung to expand.

*Hernia of the lung* is an occasional sequel of injury. It results from laceration of the muscles and parietal pleura, and appears as a rounded swelling which increases in size on coughing or on forced expiration. There is a sensation of crepitation when the swelling is palpated, and on auscultation a loud vesicular murmur may be heard. A truss or pad is all that is necessary to control the swelling as a rule. The lung may

prolapse through an open wound of the chest wall, in which case the diagnosis is obvious.

Chest injury cannot *cause* tuberculosis, although it may activate a latent focus. This point is sometimes brought up in the courts and it may have to be decided what part, if any, was played by an accident in causing or aggravating tuberculosis. There is no evidence that injury ever leads to the development of a growth.

## CHAPTER 20

### THE PULMONARY CIRCULATION

THE pulmonary circulation consists mainly of venous blood which is brought to the lungs by the pulmonary artery. The blood receives oxygen and gives off carbon dioxide during its passage through the capillaries which run between the alveoli, and it then pursues its course by way of the pulmonary veins to the left side of the heart. In addition a small supply of arterial blood is delivered to the bronchial arteries which, however, appear to play a minor part in carrying on the pulmonary circulation.

*The pulmonary artery.*—This is found to be dilated in all conditions in which there is chronic obstruction to the circulation of blood through the lungs. So far as the lungs are concerned the main causes are *hypertrophic emphysema* and diffuse fibrosis. Another common cause of dilatation of the pulmonary artery is *mitral stenosis*.

The enlargement of the artery is best appreciated on an X-ray film, which shows a projecting cone below the arch of the aorta. In some cases, when the arterial wall is weak, an aneurysm of the pulmonary artery may result. In this very rare condition there may be a pulsating swelling in the region of the second left intercostal space.

Obstruction of the pulmonary circulation by any of the above-mentioned causes leads to a rise in pressure within the pulmonary artery and may cause changes in its wall which are comparable with the changes which occur in the wall of the aorta as a result of hypertension. Atheromatous patches are often present, but they are of pathological importance only. The sole physical sign is increased loudness of the second sound at the pulmonary base, which indicates increased pressure in the pulmonary artery.

In another condition, known as *Ayerza's syndrome*, there is a diffuse sclerosis of the whole pulmonary arterial system which causes gross interference with oxygenation of the blood. It may result either from arteriosclerosis or from syphilis, and is associated with cyanosis and polycythaemia, the red cell count being in the region of 8 millions per c.mm. There is no enlargement of the

spleen, which serves to distinguish the condition from Vaquez' disease. The chief symptom is dyspnoea, and there may be some cough, sputum, and occasional haemoptysis. *Cyanosis* is often extreme and clubbing may occur. X-ray examination usually shows a prominent pulmonary cone and enlargement of the right side of the heart. The Wassermann reaction is positive in some cases. The disease is chronic and progressive, and death usually occurs after a few years from right heart-failure. Treatment is not very effective, but repeated venesection is helpful and phenyl hydrazine may be given. Syphilitic cases should receive energetic treatment.

*The pulmonary veins.*—Little attention has been paid to the pulmonary veins, yet these structures are important in view of their function of transporting aerated blood to the heart and thence to the systemic circulation. The chief pathological condition which can be recognized is sclerosis, with thickening of the intima, which occurs with chronic venous congestion. Local disease of the pulmonary veins predisposes to thrombosis and results in one type of pulmonary infarct.

*Congestion of the pulmonary circulation.*—"Congestion of the lungs" is a condition which is often wrongly diagnosed and it is important to understand the exact mechanism by which it is brought about. There are two chief forms.

*Passive congestion.*—This results from any chronic obstruction to the return of blood from the lungs to the heart. It is therefore found in mitral stenosis, and when there is chronic failure of function of the left ventricle.

The condition found post mortem is termed "brown induration". The bases of the lungs are bulky, tough, and oedematous, and the cut surface shows a brownish tint which turns red on exposure to air. Under the microscope it is seen that the capillaries are distended, there is increase of the fibrous tissue in the interalveolar septa, and there is a deposit of brown pigment in the cells of the alveoli. This pigment is derived from haemoglobin.

The symptoms are those of heart-failure, *dyspnoea*, cough with *frothy sputum*, and *haemoptysis*. The patient presents the picture of heart-failure, with cyanosis and oedema of the legs. The percussion note is impaired, the breath sounds are weak, and there are rales at the bases of the lungs. Treatment must be directed to the cardiac condition. Camphor, digitalis, strychnine, and oxygen are indicated and venesection may afford relief.



*Hypostatic congestion (hypostatic pneumonia).*—This occurs, commonly as a terminal event, in the aged and debilitated; it is nearly always present in those who have died after being for some days in a state of coma. It is also common in patients who are out of condition and who have to be immobilized for a long period of time, as may occur with a fracture of the femur or of the spine.

The condition is, as the term "hypostatic" implies, the result of failure of the pulmonary circulation, with localization by gravity to the most dependent parts of the lungs. The lower lobes are dark red in colour, solid, engorged, and pit on pressure. Portions may sink in water, and the cut surface exudes blood and serum freely. The consistency of the lung is quite different from that of chronic venous congestion, in that the lung is much softer, and the term "splenization" is sometimes employed to describe it.

The symptoms are indefinite, but *dyspnoea* and *cyanosis* are usually present. The condition is not due to infection and the patient is usually afebrile; the respirations are moderately increased. On examination of the chest there is some impairment of percussion note at the base of one or both lungs, the breath sounds are feeble, and rales are present. When the condition is advanced there may be distant bronchial breathing. As hypostatic congestion is a terminal event in the course of some other disease the prognosis is bad as a rule. It may, however, be prevented in those who are likely to develop the condition by causing the position to be changed at frequent intervals, in order that the opportunity shall not arise for any particular area of the lung to become congested. When the condition is established the only possible lines of treatment are cardiac stimulants and oxygen, although they are not likely to be very effective.

*Oedema of the lung* is caused by transudation of plasma from the capillaries into the interalveolar connective tissue and into the alveoli themselves. This is a serious condition which usually results from failure of the left ventricle. The right side of the heart continues to pump blood into the pulmonary artery and the mechanism of removal is not sufficient to keep pace with the supply. It may be acute or chronic.

(a) *Acute oedema of the lungs* was formerly considered to be due to sudden failure of the left ventricle, but it has been

shown that this view is incorrect. There is, in fact, no satisfactory explanation of this occurrence, although it is suggested that it may be due to a disturbance of the nervous control of the blood vessels in the lungs, resulting in an increase in the permeability of the capillaries. It is found in patients who have suffered for some time from hypertension, and it may occur in those who are chronically debilitated for any reason. Sometimes it complicates a major operation, but it is doubtful whether it is due to the anaesthetic. It may occur when there is a sudden alteration in the pressure in the pulmonary circulation as a result of the too rapid withdrawal of fluid from the pleural cavity during paracentesis thoracis, and it is one of the complications which should always be kept in mind when performing this operation. An idiopathic form is also described which is comparable with angioneurotic oedema.

The lungs are pale, bulky, and semi-solid, pitting on pressure. There is little congestion and on section much frothy fluid escapes.

The onset of symptoms is abrupt, with intense dyspnoea, incessant cough, and the copious expectoration of watery, frothy sputum which is sometimes blood-stained. On examination the patient is much distressed, cyanosed, pale, and sweating. The pulse is rapid and feeble and the blood pressure is low. The percussion note is usually resonant and medium rales are heard throughout the chest.

The condition is very serious and is often fatal within a few hours, but recovery may occur if energetic treatment can be instituted at once. It is customary to give an injection of  $\frac{1}{4}$  grain of morphia and  $\frac{1}{16}$  grain of atropine; this may be repeated after half an hour if the attack persists. The value of adrenalin is doubtful and, in fact, many regard it as a dangerous drug in this type of case. Strophanthin or theophyllin is sometimes given intravenously. Nitrites may relieve the milder attacks. Oxygen should be given as a routine and venesection is sometimes of value.

(b) *Chronic oedema of the lungs* is found in association with passive congestion.

*Pulmonary embolism.*—An embolus is a foreign substance, usually blood clot, which becomes arrested in some part of the circulation. There are three main types of embolus which may be arrested in the pulmonary artery: *blood clot, air, and fat.*

*Blood clot* usually reaches the lung from one of the systemic veins. The source of the clot is usually a leg vein, commonly the femoral. Embolism is therefore a complication of phlebitis; of thrombosis due to injury, or of any condition which encourages stasis of the circulation in the legs. It is not uncommon in patients who are convalescing from major operations, irrespective of the nature of the operation, and in these cases it depends not so much upon injury to a vein as upon the enforced immobility which is a consequence of the operation. Mild local infection plays a part in precipitating the thrombosis.

The condition to which the term "*pulmonary embolism*" is applied results from the breaking-off of a large piece of blood clot and its arrest in the main pulmonary artery. The clot is usually found to be several inches in length and to be coiled up in the main vessel, completely obstructing the flow of blood. Some care is necessary at post-mortem to be sure that the apparent embolus is really an ante-mortem thrombus and not a post-mortem clot. The ante-mortem clot is rather dry and may show evidence of commencing organization, it is coiled up in the artery, and has the shape of the vessel in which it was formed, whereas post-mortem clot is softer, moist, and takes the shape of the pulmonary artery.

The clinical picture of a major pulmonary embolism is characteristic. The patient is seized with sudden severe pain in the chest and extreme *dyspnoea*, *cyanosis* develops rapidly, consciousness is lost, and death occurs within a short time. On examination the signs are indefinite, being those of a major catastrophe with acute cardiac embarrassment. The physician can do nothing. Attempts have been made, by immediate operation, to remove the clot from the pulmonary artery, but it can very rarely happen that an operation is possible, and little is to be expected from this line of treatment. When the embolus is smaller, so that the obstruction affects one branch of the artery and the opposite lung is free, death may be delayed for hours or days, and the patient may even recover. The smaller the vessel involved, the more likely is recovery to occur, but the immediate prognosis should always be guarded. Sometimes the ischaemic area of the lung breaks down and massive necrosis results (p. 197). This condition is serious but not necessarily fatal.

*Infarct of the lung* results from smaller emboli, but this is

not the only way in which the condition may occur, for a similar result may be brought about by *thrombosis* in a pulmonary vessel. Blockage of a small branch of the pulmonary artery will result in the sudden cutting-off of the blood supply to a part of the lung and the capillary vessels surrounding the ischaemic area rupture, exuding blood into the affected alveoli. The same effect may be produced by thrombosis in a radicle of the pulmonary vein. The lesion heals by fibrosis and contracts, until eventually a scar is left. Should the original embolus be septic, necrosis and suppuration may occur and an abscess may result.

Under the microscope the appearance of a red infarct is characteristic. The alveoli are seen to be stuffed full of red cells, with some fibrin and comparatively few white cells. The condition is distinguished from red hepatization in lobar pneumonia by the fact that in the latter condition there is much more fibrin than red cells.

Pulmonary infarct may result from any of the causes of pulmonary embolism, phlebitis, and post-operative conditions in particular. Perhaps the commonest cause, however, is *mitral stenosis*. It is sometimes said that pulmonary infarction in mitral stenosis is due to the detachment of a clot from the right auricular appendix, but it is much more likely that, in most cases, the infarct results from thrombosis of a pulmonary vessel. The conditions in mitral stenosis, stagnation in the pulmonary circulation, and possibly mild infection in the lung, are just those which predispose to thrombosis.

The symptoms of a small pulmonary infarct may be so slight that the occurrence is overlooked, but characteristically there are *pain*, *dyspnoea*, and *haemoptysis*. There is often a slight rise in temperature for a few days. The severity of the symptoms depends upon the size and site of the infarct. There are no signs except, occasionally, pleural friction but the lesion can be seen on an X-ray. An infarct casts a round soft shadow, and it can easily be mistaken for an Assmann's focus; in the absence of other evidence it is best to repeat the X-ray in ten days, when it should be found that the shadow of an infarct has practically disappeared. When the symptoms are severe, rest in bed is essential and heat may be applied locally. An immediate resection should be done and morphia and oxygen may be given, with cardiac stimulants if there should be collapse.

When the symptoms are slight, treatment is hardly necessary, and indeed many patients with mitral stenosis are found to have had multiple infarcts without experiencing any definite symptoms. The chief danger in the milder cases is that the cause of haemoptysis may not be appreciated and the patient may be mistakenly thought to be tuberculous.

Complete rest in bed may be desirable, even with slight symptoms in the chest, when there is a primary venous thrombosis elsewhere which might give rise to further emboli.

*Septic infarcts* are commonly present in the lungs in pyaemia, and when bacterial endocarditis affects the right side of the heart. There is often no clinical evidence of their presence and the picture is that of a general infection with pyrexia. Treatment must be directed to the cause where possible, but these cases are usually fatal.

*Air embolism* occurs when a great vein is wounded, the negative tension in the vessel sucking in atmospheric air. The air reaches the right ventricle where it is churned up into froth and, when the quantity taken in is large, the circulation may be greatly impeded, or even arrested. As a rule, however, only a small quantity of air is sucked in and the patient experiences acute pain in the chest and transient *dyspnoea*. There are few or no physical signs, and treatment is symptomatic.

Air embolism can also occur during a pneumothorax refill and it is then likely to be fatal. The air gains entrance to a radicle of the pulmonary vein whence it travels rapidly to the arteries of the brain. The patient quickly becomes unconscious and may be convulsed. No treatment is likely to have the slightest effect. Careful attention to the technique of giving a refill is the best insurance against this accident.

*Fat embolism* may occur after injuries to bones. It has been assumed to be rare because it is rarely fatal, but it is now thought to be fairly common. The symptoms are usually noticed on the third day after the injury and consist of restlessness, *dyspnoea*, pallor, and cough, with frothy or blood-stained sputum; cerebral irritation is often a feature of the condition. *Droplets of fat may be found in the sputum*. There is no special treatment, but oxygen may be given if necessary.

## CHAPTER 21

### EMPHYSEMA

EMPHYSEMA is a condition in which the alveoli are dilated and their walls are atrophic. There are four main types.

1. *Hypertrophic emphysema*.—This is the type which is commonly encountered in middle age. Both lungs are equally involved as a rule. The cause is not fully understood but it seems clear that the most important factor is *loss of elasticity of the lung tissue*. Emphysema occurs at the time of life when the degenerative conditions are most common and it is recognized that there is a hereditary influence. In addition it is thought that any condition which causes forcible and more or less continuous over-distension of the alveoli is a factor in precipitating the onset of emphysema. Chronic bronchitis, bronchial asthma, and the pneumoconioses are therefore common antecedents. It is not proven that this disease occurs particularly in players of wind instruments, but it is met with in glass-blowers. Very considerable and long-continued rise of pressure is necessary in order to produce emphysema when the bronchi and lungs are healthy. Other theories, such as forced inspiration and premature ossification of the costal cartilages, have been advanced, but they are not supported by evidence.

The lungs in emphysema are voluminous, pale, and yield a peculiar soft downy sensation on palpation; they show no tendency to collapse when the chest is opened. There is pitting on pressure as a result of the loss of elasticity. A characteristic feature is the presence of bullae, which vary from a few millimetres to several centimetres in diameter. These are most commonly seen in the poorly supported parts, along the anterior margins, at the apices, and at the bases. The alveolar distension results in obstruction of the pulmonary circulation; the right side of the heart is enlarged and the pulmonary arterioles are often atheromatous. There is progressive embarrassment of the pulmonary circulation with eventual failure of the right heart. There are also changes in the chest wall. The general shape is characteristic. The ribs are more horizontal than normal and the intercostal spaces are wide. The subcostal

angle is obtuse and there is usually some degree of kyphosis. Calcification of the costal cartilages is commonly present.

Histologically the most striking change is the large size of the alveoli and the attenuated interalveolar septa. The enlargement of the alveoli results partly from actual distension and partly from disappearance of some of the septa, so that the spaces are composed of the remains of two or more of the original alveoli. The arterioles and capillaries which run in the septa are much narrowed and, in advanced cases, they may disappear completely, so that the lung is much less vascular than it should be.

Hypertrophic emphysema is usually associated with chronic bronchitis. It also develops in the course of time in chronic asthma.

*Symptoms:* The most important symptom of emphysema is *dyspnoea*. There may be cough, and a certain amount of sputum, from the associated bronchitis. Small haemoptyses may occur. When heart-failure occurs in the later stages there may be aggravation of the *dyspnoea*, and swelling of the legs.

The patient is usually highly coloured, or there may be cyanosis, which is sometimes marked and out of proportion to the *dyspnoea*.

On examination of the chest the chief feature is increase of the antero-posterior diameter, so that the transverse plane tends to become circular instead of elliptical. The presence of kyphosis may tend to mask this appearance when the patient is viewed from the front, and the degree of emphysema must always be estimated after inspection of the chest from all angles. The expansion is symmetrical and the chest appears to be constantly in a position of full inspiration. As a rule the cardiac impulse is not visible.

Movement is much impaired and the chest expansion, which should always be measured, is often less than one inch. The vocal vibrations are often a little diminished. The cardiac impulse is difficult to feel and it may be completely absent. The mediastinum is not displaced.

The percussion note is uniformly hyper-resonant and the area of cardiac dullness is diminished or even absent.

The breath sounds are weak as a rule and expiration is prolonged. There may be *rhonchi* or *sibilli* when there is

associated bronchitis. Rales may be heard at the bases when there is congestion due to heart-failure. The vital capacity (p. 16) is a reliable guide to the extent of the damage to the lungs.

The signs of this form of emphysema are very characteristic, but two things must be remembered in considering the examination of the emphysematous chest. In the first place, the condition may mask an underlying disease of the lung, such as tubercle, bronchiectasis, or growth. It is important, therefore, not to accept the physical signs at their face value, especially if there should be suspicious symptoms. Secondly, it is impossible to estimate the size of the heart with any approach to accuracy and, as the state of the right heart has an important bearing upon prognosis, special measures for the estimation of the cardiac function may need to be taken. The X-ray appearances of emphysema are characteristic. The ribs are more horizontal than usual and the intercostal spaces are widened. The lung fields are unduly translucent and bullae may sometimes be visible. The pulmonary artery may appear as a prominence in the left hilum and the right side of the heart may be enlarged. The exclusion of other disease is a most important function of the film.

Lipiodol examination usually shows that the bronchi are narrowed.

*Complications of emphysema:* Bronchitis is commonly present; a mild degree of tubular bronchiectasis may develop in the lower lobes. The only other important complication is spontaneous pneumothorax, which results from the rupture of a subpleural bulla. It must be remembered that the lung shows little tendency to collapse and that the pneumothorax may therefore be partial; the physical signs are often indefinite and a good X-ray may be necessary in order to establish the diagnosis. A relatively slight degree of collapse is sufficient to cause marked aggravation of the dyspnoea and this complication may therefore be serious.

*Treatment:* Emphysema tends to be a progressive condition and the state of the patient becomes worse winter by winter. Nothing can be done to influence the state of the lung and treatment consists in attention to the surroundings of the patient and efforts to guard against bronchitis. Any patient whose occupation involves strain on the lungs, or who is exposed



## CHAPTER 22

### COLLAPSE OF THE LUNG

THE term "collapse" indicates that part of the lung is deflated, whereas "atelectasis" means that the lung has failed to expand. Although the physical state of the lung may be identical in the two conditions it is not strictly correct to regard these terms as being synonymous.

Collapse occurs in the following conditions :—

- (a) Atelectasis, or congenital failure of expansion.
- (b) Collapse due to bronchial obstruction.
- (c) Collapse due to rise in the intrapleural pressure.
- (d) Local collapse.

In order to understand the different ways in which collapse can be brought about, it is necessary to bear in mind the normal mechanism by which the lungs are kept expanded. The pleural cavity is merely a potential space, and the lungs are constantly trying to retract, but they are unable to do so because of the tension between the two layers of the pleura. When the vacuum is destroyed, either by introduction of air or by accumulation of liquid in the pleural cavity, the lung will be separated from the chest wall and will contain less air, becoming to some extent collapsed. The process may be passive or active according to the pressure in the pleural cavity. So long as the intrapleural pressure is negative or, at most, atmospheric, the lung retracts spontaneously because of the elastic tissue which it contains, but, when the pressure in the pleural cavity becomes positive, there is a fresh factor of active compression of the lung, with furtherance of the collapse process.

The lung may become airless for an entirely different reason. Complete occlusion of a bronchus results in the cessation of the air supply to the affected part, and in the absorption of the air already contained in the lung into the blood stream. The result, so far as the lung tissue is concerned, is exactly the same as in the first case, but, as the pleural cavity is not affected, there is no possibility of retraction of the lung away from the chest wall. The space which is left as a result of the contraction

of the lung must be filled up in some way, and this is effected by contraction of the chest wall, shift of the mediastinum towards the affected side, expansion of the opposite lung, and rise of the diaphragm.

(a) *Atelectasis*.—The foetal lung is a solid structure which microscopically resembles a secreting gland. It is only after the child is born, and after the first few breaths have been taken, that the lung expands and begins to assume the appearance with which we are familiar. It sometimes happens that a lobe, for some reason that we do not fully understand, fails to expand in the normal manner. It appears always to be a lower lobe, and usually on the left side. The only result at the time is that the remainder of the lung on the affected side is over-expanded in order to compensate for the deficiency. There are no symptoms and, in most cases, no abnormal physical signs to be detected, although sometimes the breath sounds are found to be a little weak at the affected base.

Occasionally tubercle or bronchiectasis occurs in a lobe which has failed to expand, and cough, with sputum, and even haemoptysis, may draw attention to the abnormality. When the collapsed lobe is diseased there may be bronchial breathing and local rales.

The diagnosis can be made with certainty on the X-ray, which shows a characteristic triangular shadow. When the lesion is behind the heart this shadow may be overlooked on superficial examination of the film (fig. 21), but attention is drawn to it by the fact that the lung tissue in the left costophrenic sinus is more translucent than that on the opposite side, as a result of compensatory expansion of the upper lobe. The presence of such an area of emphysema should always lead to careful examination of the region behind the heart.

When the middle lobe is shrunk, either by collapse or by fibrosis, the physical signs are indefinite and an X-ray is essential. The antero-posterior view is not conclusive, but the lateral view shows a triangular shadow projecting forwards across the middle of the film.

A lipiodol examination may demonstrate bronchiectasis or an obstructed bronchus. A similar condition may occur as a transient event when the bronchus is blocked by secretion, and it is impossible to be sure whether the shadow represents a true atelectatic lobe until the patient has been under observation,

and until it is certain that the lobe will not re-expand. An exactly similar radiological appearance may result when a lower bronchus is obstructed by growth. Bronchoscopy is usually necessary in order to be sure of the cause of the obstruction.

(b) *Collapse due to bronchial obstruction*.—A lobe, or a complete lung, may be collapsed as a result of obstruction of the bronchus which supplies it, and the process may occur suddenly or gradually.

(i) *Acute collapse of the lung (massive collapse)*.—This condition was recognized for the first time as recently as 1908, and the mechanism by which it is brought about has only been properly understood since bronchoscopy became a practical method of investigation. When a large bronchus is completely obstructed the air in the lung becomes absorbed into the blood stream. When the obstruction occurs suddenly it can be shown by serial skiagrams that the lung becomes completely deflated in from six to ten hours. The acute condition is most commonly seen following major operations, especially upon the abdomen, and it must then be carefully distinguished from true post-operative pneumonia. It may also occur in bronchitis and asthma, following inhalation of a foreign body, and rarely as a complication of gun-shot wounds in the chest or upper abdomen.

The causation of acute massive collapse has been much discussed, and at one time it was considered to result from interference with the action of the diaphragm. It is known, however, that diaphragmatic paralysis, following phrenic avulsion, hardly ever results in massive collapse of the lung, and, since the introduction of bronchoscopy as a routine method of investigation, it has been found that practically all cases of spontaneous collapse result from bronchial obstruction, usually by a plug of muco-pus. Collapse may occasionally result from the aspiration of blood or other liquid, and it has occurred as a sequel to giving lipiodol for diagnostic purposes. Post-operative bronchial obstruction often results from pre-operative medication; atropine renders the bronchial secretion dry and tenacious, and morphia depresses the cough reflex. Hence the plug.

The clinical picture of acute spontaneous collapse resembles that of pneumonia, but the correct diagnosis can be made on the physical signs. The severity of the symptoms depends upon

the rate at which the collapse occurs. The more sudden the development the more closely the clinical picture resembles that of pneumonia. There is often a rise of temperature of two or three degrees, the pulse rate is raised, but the respiration rate is either unaltered or, at any rate, not much increased. This point is of some importance for it helps to distinguish the two conditions.

The symptoms are *paroxysmal cough*, with little or no sputum, and pain in the affected side of the chest. This pain is very common and it appears to result from sudden increase in the negative tension in the pleural space when the lung is becoming deflated. *Dyspnoea* is often intense at the onset but becomes gradually less.

The physical signs are pathognomonic.

On inspection the patient appears ill and cyanosed. The chest is asymmetrical, and the affected side is retracted and almost immobile. The cardiac impulse can often be seen to be displaced towards the airless side.

On palpation the shift of the mediastinal structures towards the affected side is confirmed; the vocal vibrations are usually diminished.

The percussion note is much impaired, especially in comparison with the somewhat hyper-resonant note on the opposite side. When one lobe only is affected the resonance over the other lobe on the same side may be increased.

The chief physical sign on auscultation is the *absence of breath sounds*. This is the characteristic sign of bronchial obstruction, and it serves to distinguish collapse of the lung from fibrosis. There are no added sounds as a rule, although crepitations may be heard when the collapse is incomplete.

When the condition occurs as a complication of bronchitis and asthma it may affect either a lobe or a whole lung. In cases of post-operative massive collapse it is nearly always a lower lobe, more commonly the right, which is affected. The diagnosis from pneumonia is made by the fact that the patient is not so toxic, the respirations are not so rapid, and, on physical examination, the breath sounds are found to be weak or absent, while the *mediastinum* is displaced towards the affected side. The X-ray appearances are conclusive (figs. 13, 22).

The only complication which is important is infection of the collapsed lung. This may give rise to a pneumonitis or to lung abscess, but expansion usually occurs spontaneously and the risk of suppuration is not very great.

*Treatment* should preferably be prophylactic. If a part of the lung shows a tendency to collapse, as is indicated by weakening breath sounds, the patient should be encouraged to breathe deeply. Sometimes this is difficult, especially when an abdominal operation has recently been performed, and in such cases the inhalation of 5 per cent carbon dioxide may assist in increasing the pulmonary ventilation and thus may help to prevent collapse. A powerful respiratory stimulant is coramine; the intravenous injection of 7 c.cms. of this drug may cause sufficient cough to cause expulsion of the plug. When the condition has already occurred it is unwise to encourage deep breathing, for the only effect which this could produce would be to drive infection deeper into the bronchial tract. It is wiser to wait for a few days in the hope that the obstruction will clear itself, as is often the case. If the patient does not show signs of improvement, or if the temperature persists, indicating the possibility of commencing suppuration, a bronchoscope should be passed in order to clear the obstructed bronchus.

(ii) *Chronic massive collapse*.—When the bronchus is gradually occluded there may be no definite symptoms and the patient may be quite unaware that one lung is out of action. Such symptoms as there are will be referable to the cause of the obstruction.

The causes of bronchial obstruction are :—

*Intrinsic.*

Bronchial tumour, malignant or innocent.

Foreign body.

Inflammatory stricture (rare).

*Extrinsic.*

Pressure on the bronchus. The chief causes are aneurysm, malignant glands and, rarely, tuberculous glands.

The condition should be easily recognized on physical examination. On inspection there is retraction and immobility of the chest, and the cardiac impulse is displaced towards the affected side.

The vocal vibrations are diminished; the shift of the mediastinum can be confirmed. The axillae should be examined for glands.

The percussion note is much impaired, or even dull.

The breath sounds are absent.

Careful investigation of these cases is most important. The sputum must be examined as a routine.

The X-ray film shows characteristic changes. When the whole

lung is collapsed the field is uniformly opaque, and the density is equal to that due to fluid. When the collapse is on the left side the right border of the heart may be completely invisible, for the heart is drawn into an area of similar density. When the right lung is collapsed the actual displacement and situation of the heart can be made out. The shift of the trachea can usually be clearly seen (fig. 13).

When a lobe is collapsed the resulting shadow is usually triangular, with its base towards the mediastinum (figs. 21, 22). The shadow is uniformly dense and the outer border is sharply defined. Displacement of the mediastinum may not occur, for the remaining lobe on the affected side may enlarge to fill the space. When the middle lobe is collapsed the shadow is seen as a small triangular area in the lower part of the right hilum. Sometimes this shadow may closely simulate a contracted right lower lobe, but a lateral view will differentiate the two conditions at once. Collapse of the middle lobe throws a shadow which projects forwards, whereas collapse of the lower lobe is seen as a dense area in the lower and posterior part of the chest.

Lipiodol examination is valuable in locating the exact site of the obstruction.

A routine bronchoscopy should be done in every case in which there is collapse of a major portion of the lung, partly for diagnosis, and partly in order to decide whether local treatment is likely to be effective. A foreign body or an innocent tumour may be removed, or material can be obtained for microscopic examination.

(iii) *Collapse due to rise in the intrapleural pressure.*—The condition causing the collapse is usually a pneumothorax or a pleural effusion. Less commonly aneurysm, a large tumour, or diaphragmatic hernia may produce a similar result. The symptoms and signs are always those of the condition which causes the lung to become deflated. The degree of dyspnoea is proportionate to the rate at which the lung is put out of action.

The collapse may be partial or complete. When the pleura is free the lung retracts of its own accord until it forms a small spherical mass in the hilum, but an emphysematous lung does not collapse nearly so completely. When there are adhesions between the layers of the pleura, collapse is only partial. A diseased lobe usually collapses more easily than a normal one.

The treatment of this condition is that of the cause.

(iv) *Local collapse.*—Small areas of collapse occur in many acute conditions such as bronchopneumonia, whooping-cough,

and asthma. They may also be found around tuberculous areas and in chronic cachectic states.

The symptoms are those of the causal condition. There is only one physical sign of this type of collapse and that is the presence of crepitations which clear on coughing. These always indicate involvement of the parenchyma of the lung.

The condition is often seen at post-mortem, when inspection shows small depressed areas of a greyish colour, well defined, and firm. On section the area is usually triangular and the airless tissue sinks in water.

The condition is a pathological rather than a clinical entity and the treatment is that of the cause.

### *Post-operative Chest Complications*

Respiratory complications frequently follow operations, especially on the abdomen. It has been shown that smokers are much more liable to these misadventures, especially bronchitis and collapse of the lung. It is a wise precaution to abandon the use of tobacco for three weeks before a major operation.

1. *Acute bronchitis*.—This is common after a general anaesthetic, especially ether. It can be guarded against by careful selection of the anaesthetic and by taking precautions to avoid exposure during transit from the ward to the theatre.

2. *Pneumonia*.—This is rare and usually occurs as bronchopneumonia. It may follow either general or spinal anaesthesia. Although it is commoner in patients with a history of previous respiratory disease, it is a rather frequent sequel of operations on the upper respiratory tract. A useful method of prevention is to give a course of sulphonamide, commencing on the day before the operation is due to take place.

3. *Lung abscess*.—This complication follows operations on the upper respiratory tract or on the abdomen. It is fully dealt with elsewhere (p. 197).

4. *Massive collapse of the lung*.—This is a frequent sequel of abdominal operations. The cause is obstruction of a bronchus by a plug of mucus, or mucus-pus. It is thought to be due to the depression of the cough reflex which occurs after morphia. There is evidence to suggest, however, that atropin given before the anaesthetic renders secretion sticky and difficult to dislodge, and its use is therefore not wholly desirable as a pre-operative measure.

5. *Oedema of the lungs*.—This results from left ventricular failure and the prognosis is bad. It is impossible to guard against this complication.

6. *Pulmonary embolism*.—This is commonest after abdominal operations and usually occurs about the tenth day. When the

embolus is large there may be sudden death. Smaller emboli frequently occur causing pulmonary infarction. This is not particularly serious unless multiple infected emboli lodge in the lungs as part of a pyaemia.

7. *Pleural effusion*.—This nearly always results from direct spread from infection in the lung. Clear pleural effusion may occur after an infarct. Empyema follows pneumonia or septic infarct. Occasionally there appears to be a direct spread from an infection under the diaphragm. This effusion may remain clear, but an empyema may develop.

8. It may also be remembered that any operation, but especially when a general anaesthetic is given, may reactivate previous quiescent disease in the lungs. This applies particularly to patients with latent, or presumably quiescent, tuberculosis. A careful examination of the lungs, preferably with an X-ray, should always be carried out before a general anaesthetic is given.



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## CHAPTER 23

### PNEUMONIA

THE term "pneumonia" is used in a general sense to indicate acute inflammation of the lung. Several distinct types can be recognized, but these tend to merge into each other so that atypical forms are often encountered.

#### Lobar Pneumonia

This is an acute specific infection which, in its typical form, is a very characteristic disease, *self-limiting*, and usually terminating by a crisis. The essential feature of true lobar pneumonia is *dyspnoea with a markedly raised respiration rate*. That this is not in any way dependent upon the degree of lung involvement is shown by the fact that an abrupt increase in the respiration rate coincides with the onset of the pneumonia and it is not in any way proportional to the extent of consolidation. In fact the only possible explanation of this characteristic symptom is that the infection gives rise to a toxæmia which has a specific influence upon the respiratory centre. It is uncertain whether this toxic effect depends upon the presence of a particular type of organism, whether it is simply a question of the number of organisms present, or whether it results from some breakdown of the resistance mechanism of the patient. In any case, the result is characteristic and the presence of this symptom conveys information of the utmost diagnostic and prognostic value.

*Ætiology.*—Lobar pneumonia is quite common in young children, in whom it tends to run a comparatively mild course, but it is less prevalent in older children. After puberty the incidence and the mortality both increase rather rapidly until middle age, by which period pneumonia is one of the chief causes of death. It is three times as common in males as in females. The disease is not very infectious, although there is evidence that it can be transmitted. It tends to occur in the winter months and it may appear in epidemic form. Anyone may be attacked and the disease does not spare those who

appear to be in good health, although those whose resistance to infection is lowered are naturally more liable to be affected. Pneumonia may follow an upper respiratory infection, such as a neglected "cold" or influenza. Fatigue and exposure to cold are thought to be factors in some cases. Chest injuries rarely, if ever, cause it, and it is not an occupational disease.

Lobar pneumonia is a frequent terminal event in chronic diseases such as cirrhosis of the liver, diabetes, chronic nephritis, the severe anaemias, chronic nervous diseases, and cancer. It is a common cause of death in the aged.

The immunity conferred by an attack is short-lived and it may even happen that the resistance of the patient is actually lessened, and thus further attacks may occur. Factors which lower the general resistance, such as alcoholism, predispose to pneumonia and also modify the prognosis.

*Pathology.*—Although the organism first isolated from cases of lobar pneumonia was Friedlander's bacillus, it is now recognized that the condition is nearly always due to infection with the pneumococcus. There are at least 32 serological types, of which types 1, 2, and 3 are by far the most virulent. Pneumococci are frequently found in the upper respiratory tract of healthy persons, but types 1 and 2 are rarely found in the mouths of normal people. It would seem that the less virulent organisms are able to maintain themselves for long periods in the nasal mucosa without causing acute disease, whereas the really virulent types either produce an acute infection or are destroyed.

The physical state of the host is equally important in determining the onset of pneumonia after infection with a virulent organism. Those who are out of health, who suffer from a debilitating disease, or who neglect an apparently minor infection, are more liable to contract the disease. Nevertheless a sufficiently virulent pneumococcus will cause pneumonia in a robust person. There is evidence that a temporary event, such as exposure to cold, can lower resistance sufficiently to allow the infection to gain the necessary foothold.

The pneumococci reach the lung through the bronchi. There is less certainty about the initial stages of the inflammation. Recent experimental work suggests that the initial lesion is often peripheral, and that the spread of the infection is due to the passage of fluid, heavily charged with pneumococci, from

each alveolus to the next and also to the wider diffusion of this fluid along the bronchioles.

Certain very curious immunological factors appear also to be concerned. It seems that pneumonia does not occur in those whose blood is lacking immune bodies to the pneumococcus, in fact the development of the disease apparently requires a pre-existing humoral immunity; in early cases of pneumonia the capacity of the blood for killing the pneumococcus is said to be high.

It is also sometimes contended that the early changes in lobar pneumonia are really allergic. The evidence bearing upon this is at present conflicting.

The changes in lobar pneumonia are analogous to those which occur in any other acute inflammation, but modified by the nature of the tissue involved. The process is progressive and therefore different areas of the lung will show different stages of inflammation in the earlier phases but four classical stages are described.

(a) *Congestion*.—The inter-alveolar capillaries dilate and plasma is poured out into the alveoli. The lung appears congested but it is not yet solid.

(b) *Red hepatization*.—The plasma in the alveoli clots, so that the spaces are firmly plugged with fibrin. The circulation persists but oxygen cannot reach the blood; this "shunt" is an important factor in causing cyanosis (p. 30). The lobe is bulky, solid, and sinks in water. The pleura loses its lustre. On section, the lobe cuts with a sense of resistance and the cut surface is deep red in colour and dry. Under the microscope the alveoli are seen to be full of fibrinous exudate and the capillaries which run between the alveoli are greatly dilated. It is this dilatation of the capillaries which causes the red appearance which gives the name to this stage of the disease, and it must be noted that there is little diapedesis of red cells into the alveoli. It is a common mistake to diagnose a section which shows alveoli stuffed with red cells as red hepatization in lobar pneumonia, but this appearance is characteristic of red infarct. Red hepatization is rarely seen in the post-mortem room, because it is a very transient stage and a fatal result does not usually occur so early.

(c) *Grey hepatization*.—Quite soon after consolidation has occurred the next stage begins. It is all a part of the acute in-

flammatory process and consists in a migration of polymorpho-nuclears from the capillaries into the exudate in the alveoli. There is thus a gradual transition from red to grey. The lobe is still bulky but less dry ; it cuts with resistance, and the cut surface now shows a uniform yellowish grey colour. The pleura overlying the inflamed area is covered with fibrinous exudate. The regional lymphatic glands are enlarged and congested. Under the microscope it is seen that the capillaries now contain little blood and that the exudate in the alveoli consists largely of leucocytes. As the process goes on the number of white cells increases until the alveoli are full of what is, for all practical purposes, pus.

(d) *Resolution*.—In the final stage the inflammatory products are dispersed. Part of the material finds its way into the bronchi and is coughed up, part is absorbed by the lymphatics, and the remainder by the blood. In the majority of cases the inflammatory material is completely removed and the lung returns to its normal state. The fibrinous pleurisy which so commonly accompanies lobar pneumonia cannot be repaired quite so completely, and it is usual to find that pleural adhesions are left.

### *The Clinical Picture*

The classical story of lobar pneumonia must still be told, although the universal use of the sulphonamides and penicillin has reduced the traditional picture to the status of a rarity. Few students in future will have the opportunity of seeing a natural crisis on the seventh day of the disease.

*The onset*.—The onset of symptoms is sudden, with a rigor or even, in children, with a convulsion. The appearance of the patient suggests an acute infection, for there is fever, sweating, anorexia, constipation, and severe malaise. A patch of herpes on the lips is very common. Dyspnoea and rapid respiration rate occur almost at once and cough commonly develops within a few hours. On examination the patient is flushed, the skin is moist, and the tongue is coated with white fur. The temperature, pulse, and respiration rate are all raised. In the early stages there may be no physical signs of local respiratory disease but, when exudation is commencing, there is a phase in which *indurated crepitations* are heard. These added sounds are an indication of a lesion which involves the lung parenchyma.

## 176 DISEASES OF THE RESPIRATORY TRACT

In cases in which the initial changes are peripheral, pleurisy may occur at an early stage, causing *pain on inspiration*, and *pleural friction*.

The stage of onset usually lasts for one or two days until the development of the disease produces the typical picture of pneumonia.

*The course.*—The signs of consolidation appear on the second or third day. The symptoms and signs are now largely referable to the chest. The febrile symptoms are even more marked, cough is often troublesome and painful, and *sputum* appears; mucoid at first, it rapidly assumes the typical "rusty" character as a result of the presence of a little altered blood. Frank haemoptysis is rare. Dyspnoea increases and may cause much distress.

The patient is often cyanosed at this stage. Physical signs of consolidation are usually very definite. The affected side may appear to be a little prominent and movement is diminished. *Vocal vibrations are increased*. There is no displacement of the heart, but it must be noted that the toxic process may cause the ventricles to dilate and the heart may therefore be enlarged. The percussion note is impaired, but not usually dull. The shape of the area of dullness is of some help in distinguishing between a solid lower lobe and a pleural effusion, for consolidation causes the line of demarcation between the normal and the solid lobes to run downwards and forwards from the spine of the scapula through the axilla to the front of the chest, whereas, with fluid, the area of dullness rises from the spinal column towards the axilla and then falls towards the sternum. The breath sounds are loud and bronchial, and as a rule there are no added sounds, except possibly pleural friction. The voice sounds are bronchophonic. Should the bronchus become blocked with secretion the breath sounds will disappear and the distinction between consolidation, effusion, and collapse may be difficult. The urine is scanty and highly coloured, with a heavy deposit of urates; the excretion of chloride is diminished.

In the patient who is doing well the condition, both symptoms and signs, remains stationary for several days. The temperature remains steady between 102° and 103°, the pulse about 110, and the respirations about 40 per minute. Sweating is fairly common and insomnia may be troublesome.

## PNEUMONIA

*The termination.*—Lobar pneumonia is a self-limiting disease, and the most common termination is by *crisis*. This occurs in the untreated patient when the immunity mechanism has been fully brought into play; there is an abrupt fall in the temperature and pulse and, to a less extent, in the respirations. The general condition of the patient improves at the same time. The crisis occurs most commonly upon the seventh day of the disease, but may occur at any time between the fifth and ninth days. The temperature does not always fall in this way but sometimes by *lysis*; in these cases it falls one or two degrees each day for two or three days, and the improvement in the general condition is correspondingly less dramatic. Sometimes there may be a *pseudo-crisis*, in which the temperature falls exactly as if a normal crisis has occurred, and then rises again after a few hours. This is usually followed by *lysis*. Occasionally the temperature persists, or falls very gradually over a period of several weeks. This is sometimes due to *delayed resolution*, the cause of which is not understood, but a similarly persistent temperature may result from some complication, such as empyema or lung abscess, or it may be that the condition has a tuberculous basis. The sputum must always be examined for tubercle bacilli when the temperature does not pursue its expected course.

The clinical picture in the stage of resolution is one of rapid improvement in the general condition. Cough is productive and the sputum is frankly purulent for a few days, after which these symptoms disappear. The physical signs indicate gradual resolution of the solid area, and the appearance of *reduced crepitations* indicates loosening of the inflammatory mass. The bronchial breathing becomes less intense and is gradually replaced by normal vesicular breath sounds. Showers of rales are heard for a few days and the breath sounds gradually recover their normal intensity.

*Prognosis.*—The advent of the sulphonamide group of drugs has materially improved the outlook, especially in the younger age-groups, and there is every likelihood that the results of treatment will improve still further as more effective compounds are introduced. The prognosis in lobar pneumonia depends upon the resistance of the patient and on the virulence of the infection. Those whose general condition is good obviously stand a better chance than those who suffer from wasting



## 178 DISEASES OF THE RESPIRATORY TRACT

disease or from anaemia or diabetes. The *habits* must be reviewed, for alcoholics do not do well. The state of the lungs is important in so far as previous chronic disease, such as bronchitis and emphysema, fibrosis or tubercle, make the outlook less good. The strain on the circulation is considerable and a weakened heart muscle may fail to hold out until the time of the crisis. Pneumonia due to infection with *B. Friedlander* has a much higher mortality than the pneumococcal types.

The prognosis may best be judged by the appearance of the patient. Delirium, vomiting, marked cyanosis, and a dry mouth are bad signs. Information may be gained from other observations. A temperature which is too high, for instance over  $104^{\circ}$ , indicates a severe infection, whereas a feeble temperature response indicates poor resistance. A falling blood pressure, especially when the pulse rate is rising, is a bad sign. The white-cell count should be moderately raised, and failure of leucocyte response means failure of the immunity mechanism.

The common cause of death in lobar pneumonia is heart-failure due to toxæmia or, less commonly, to mechanical embarrassment when a large area of the lung is involved. Certain of the complications may also be lethal.

### *Complications*

1. *Complications in the lung.*—Perhaps the most important local complication is *suppuration*, with consequent abscess formation. This occurs in debilitated patients and especially in diabetics. When it occurs, the temperature remains raised and there is no apparent crisis. The sputum increases in quantity and consists of pure pus. Persistent bronchial breathing and rales are the chief signs. An X-ray should be taken when the condition does not resolve as it should, and this may show a cavity with a fluid level. The treatment is similar to that of primary lung abscess.

*Delayed resolution* is another fairly frequent occurrence. Cough, sputum, and temperature continue, and the signs in the chest persist over a period of weeks. It is possible for resolution to occur even when the signs have persisted for a month, but sometimes a perceptible degree of pulmonary fibrosis remains; bronchiectasis is a rare sequel. It is sometimes possible to provoke resolution by needling the solid lung. This may be

done inadvertently when exploring the chest for fluid, with beneficial result to the patient.

Pulmonary tuberculosis is not strictly a complication nor a sequel of lobar pneumonia. When tubercle is found it is safe to assume that the condition has been tuberculous from the start or that a quiescent lesion has been activated.

2. *Complications in the pleura.*—Fibrinous pleurisy is almost invariable and can therefore hardly be reckoned as a complication.

Pleural effusion occurs in a considerable number of cases, but the fluid quite often remains clear or is perhaps slightly turbid; pneumococci may, or may not, be recovered from this fluid. Should the condition of the patient be satisfactory, and the temperature be following its normal course, it is not necessary to remove the fluid.

A more serious, although less common, complication is *empyema*. The fluid may be purulent from the start, or it may become so in the course of a few days. The presence of an empyema should always be suspected if, after the crisis, the temperature rises again within a few days and becomes hectic, or if the temperature commences to swing before the crisis has occurred. Empyema is considered in a later section (p 283).

3. Most of the other complications are either toxic or septicæmic in origin.

(a) *Cardio-vascular complications.*—In severe cases the toxæmia is sufficient to produce changes in the heart muscle. There is always cloudy swelling and fatty degeneration of the muscle fibres, and clinically there is some degree of dilatation of the heart. These changes are toxic in origin. Disturbances of rhythm, premature beats, fibrillation, and heart-block are evidence of more serious damage to the heart muscle.

*Pericarditis* is rare but it has a high mortality. The onset is insidious, for the patient is so ill with the pneumonia that the symptoms, precordial pain and additional shortness of breath, may not attract attention. In any patient who is more ill than can be explained by the extent of the pneumonia, the possibility of pericarditis should be considered. Physical examination is apt to be negative, for the exudate in these cases is usually so soft that pericardial friction may be absent. When present, it may be confused with pleural friction at times. There is not usually any gross effusion in the pericardial sac.

*Infective endocarditis* may result from *pneumococcal* infection. It occurs on valves which have been previously damaged, and usually in the right side of the heart. Pneumonia is a serious event when there is congenital heart disease, for right-sided endocarditis is a distinct possibility. In these cases the patient is very ill, the cardiac signs change rapidly, pulmonary infarcts occur, and a blood culture shows the presence of pneumococci.

*Venous thrombosis* is fairly common, usually in the large veins. The blood is viscid and is very apt to clot. It is important to remember this fact when contemplating a venesection in pneumonia, for it is by no means easy to withdraw the blood because of this liability to clot formation in the needle, and the open method may be necessary.

(b) *Complications in the nervous system.*—Peripheral neuritis, toxic in origin, is fairly common; the knee jerks are often lost in the acute stage.

*Meningeal complications* are occasionally encountered, especially in children. There are two main types.

(i) *Meningism* occurs chiefly with apical pneumonia. The child complains of headache, and there is head retraction and stiffness of the neck muscles. The condition may closely simulate meningitis. The prognosis in meningism is quite good and therefore steps should be taken to make certain of the diagnosis as soon as the symptoms appear. A lumbar puncture will show clear cerebrospinal fluid with not more than a few additional cells, which are often lymphocytes, and no organisms. Recovery is always complete and the prognosis is that of the lung condition.

(ii) *Pneumococcal meningitis* is an almost invariably fatal complication. The patient is more ill than would be expected from the lung condition, there is drowsiness and severe headache but, as the vertical region is chiefly involved, head retraction may not be present. In fact, meningism often produces a more definite picture of "meningitis" than does true pneumococcal meningitis. Lumbar puncture yields fluid which may be clear or slightly turbid, and pneumococci will be found; in fulminating cases the only abnormality may be the presence of the organisms. The advent of chemotherapy may render this complication less lethal than it was formerly.

(c) *Digestive complications.*

(i) *Acute parotitis* may occur unless the hygiene of the mouth is carefully tended.

(ii) *Acute dilatation of the stomach* sometimes occurs in cases with severe toxæmia. There is persistent vomiting and the upper abdomen is greatly distended. The prognosis is bad.

(iii) *Acute hepatitis* may occur as a result of toxæmia. A slight degree of jaundice is not uncommon, but severe and progressive jaundice may occur, and in such cases there is a high mortality.

(iv) There may be *meteorism* (distension of the small bowel), which may cause discomfort and may aggravate the dyspnoea. This appears to occur chiefly in patients who have been injudiciously fed on milk.

(v) Constipation is the rule, but *diarrhoea* may occur when there is marked toxæmia.

(vi) Secondary peritonitis is rare and has a high mortality. The clinical picture is that of abdominal distension, high fever, and profound general debility.

(d) *The joints.*

Multiple arthritis sometimes occurs in children but, in the majority of cases, subsides without trouble. In severe cases suppuration may occur in one or more joints.

*Differential diagnosis.*—Typical lobar pneumonia is easy to recognize. Acute tuberculous pneumonia may simulate lobar pneumonia occasionally, and this possibility should be suspected when fever persists for more than 12 days. It is a good precaution always to have the bacteriology of the sputum fully determined, for it is important to make the diagnosis of tuberculous pneumonia as early as possible. The prognosis is more serious than in lobar pneumonia and it may be necessary to collapse the affected lung.

The physical signs sometimes cause difficulty in the distinction between consolidation and pleural effusion, either clear or purulent. With effusion the vocal vibrations are typically absent, the upper limit of the percussion dullness slopes upwards and forwards from the spine instead of downwards and outwards as occurs with a consolidated lower lobe; ægophony is suggestive of fluid. The presence of rales or crepitations is generally considered to point to consolidation rather than to effusion. An area of friction obviously excludes the possibility of fluid in

## 182 DISEASES OF THE RESPIRATORY TRACT

that region. With large effusions the heart and trachea are displaced away from the affected side.

The physical signs of massive collapse of the lung may simulate those of lobar consolidation, but the heart is displaced towards the affected side and the breath sounds are weak or absent. "*Pneumonia*" is often diagnosed after abdominal operations when the condition is in reality massive collapse.

In the first few days the clinical picture may be that of pyrexia of uncertain origin. Sometimes the pulmonary signs do not appear until the fifth day, or even later, and the common causes of pyrexia have to be considered. Occasionally the patient with pneumonia sinks into a typhoidal state and the serum agglutinations may be necessary in order to distinguish the condition from typhoid fever.

The distinction from bronchopneumonia is not always easy, but the signs are much more scattered in this condition and the course of the illness does not show the uniformity of lobar pneumonia.

Primary lung abscess may simulate pneumonia but the sputum is rarely rusty, being more likely to contain frank blood at first, and later pus. The temperature may be as high as in lobar pneumonia but the respirations are little raised; it may be noticed that change of position causes cough. An X-ray may be necessary to settle the diagnosis.

Basal pneumonia is often associated with diaphragmatic pleurisy which may cause such severe abdominal pain as to stimulate an abdominal emergency. There may be vomiting and muscular rigidity, and the picture may closely resemble that of acute appendicitis or perforated gastric ulcer. The possibility of diaphragmatic pleurisy should be kept in mind in apparent acute abdominal conditions, particularly when there are atypical features.

*Treatment.*—Penicillin and the sulphonamides have entirely altered the prognosis and treatment of acute chest infections. It must not be thought, however, that treatment begins and ends with drugs. Careful management and good nursing are as important as ever. The patient must be confined strictly to bed, in whichever position is most comfortable. There must be free ventilation and the temperature should be kept constant at about 60° F. Treatment in the open air has been suggested, and in some countries the climate is, no doubt, suitable for this.

The diet should be entirely liquid, and it must be remembered that the digestive tract in the febrile stage is not capable of dealing with complicated foods. Bland fluids, such as water, barley water, lemon, orange, and grape-fruit drinks, should be given in as large quantities as the patient can take. Milk, preferably citrated, may be given in *small* quantities and it is unwise to give more than one pint in 24 hours. It is usually found that patients who are given much milk have consequent abdominal discomfort, flatulence, and distension. Eggs and cereal foods may be added if the patient so desires.

Attention to the bowels is probably overdone in most cases. The simplest laxatives are preferable, and enemata are best of all. Abdominal distension may be treated by a turpentine enema or a turpentine stupe, or pituitrin may be injected subcutaneously.

Specific treatment should be started without delay. At present the choice lies between the sulphonamides and penicillin. From the point of view of efficiency there is little to choose between these two when the infection is due to the pneumococcus, but the convenience of the oral administration of tablets, as opposed to frequent injections, usually determines the choice of a sulphonamide in the beginning.

The original drugs in this series, such as sulphanilamide and sulphapyridine, have now been superseded by compounds which are at least as effective and much less toxic. Cyanosis hardly ever results from the use of the latest derivatives, and digestive disturbances, although they do occur, are rarely troublesome. It is advisable to keep a careful watch on the leucocyte count during treatment and to discontinue the drug if the white cells fall to a level which could be termed a leucopenia. The important feature is a *falling* white count, for it appears that a low count before treatment is commenced is not necessarily a contra-indication to sulphonamide therapy. Four of the drugs in this series are in common use at the present time.

*Sulphathiazole* is effective in pneumococcal and staphylococcal infections. The initial dose is 3 or 4 gm., and the total course should consist of 30 gm. administered in four or five days. One objection to the use of sulphathiazole is that it is relatively insoluble and likely to be precipitated in the kidneys, with resultant haematuria and even possibly anuria. Plenty of fluid should therefore be given throughout the course.

*Sulphadiazine* is better tolerated than sulphathiazole and it is equally effective in pneumococcal and staphylococcal cases. It is also worth a trial in infections with *B. Friedlander*. The initial dose is 3 or 4 gm., and the total course consists of about 30 grammes.

*Sulphamezathine* is less insoluble than sulphathiazole and there is no tendency to precipitation in the kidneys. It is most useful in infections due to the pneumococcus and the haemolytic streptococcus. The initial dose is 4 gm., followed by 2 gm. six-hourly throughout the 24 hours.

*Sulfamerazine* is also used in pneumococcal and haemolytic streptococcal infections. The initial dose is 4 gm., and the maintenance dose is 1 gm. eight-hourly or 0.5 gm. every 4 hours.

Occasional cases are seen in which the administration of these drugs seems actually to increase the fever. This is an indication to stop the drug. Whenever the temperature fails to respond after 48 hours it may be assumed that the organism concerned is drug resistant; it is unlikely that any other member of the group will prove effective in cases of this sort.

Penicillin is the most efficient substance yet discovered for the treatment of pneumonia. The great majority of the organisms are penicillin sensitive and the fact can be readily established in any given case by a laboratory test (p. 70). But the drug must be given by injection, preferably at frequent intervals, and this may be inconvenient unless the patient is in hospital or unless the nursing facilities in the home are unusually good. It is a common practice, therefore, to commence treatment with one of the sulphonamides unless the illness appears to be unusually severe or there is some contra-indication to sulpha-therapy, such as chronic heart or kidney disease. In all cases of urgency, and whenever the sulphonamides are contra-indicated, penicillin should be given at once.

The most satisfactory results are obtained by giving between 20,000 and 40,000 units of penicillin by intramuscular injection at three-hourly intervals, so that the total daily dosage varies from 160,000 to 320,000 units and the blood concentration shows little variation. The frequently repeated injections are likely to become irksome to the patient after two or three days and it is now not unusual for patients to

## PNEUMONIA

be given only two injections in the day of a large dose, from 100,000 to 200,000 units of penicillin, dissolved either in oil or in water. Good results are claimed for this simplification of the standard treatment and it certainly presents some great advantages, if it can be conclusively shown that it is equally efficient. It is as yet too soon to be sure of this.

The effect of penicillin in lobar pneumonia is obtained quickly and with certainty in most cases, the mortality of the disease has been most favourably influenced, and the classical crisis can now be anticipated by three or four days. There are no contra-indications to the use of penicillin in pneumonia.

As the disease is usually due to a single organism, and not to a mixed infection, there does not appear to be any reason to combine penicillin with sulphonamido therapy in lobar pneumonia, although such a combination may well prove to be beneficial in other acute lung infections in which some of the organisms present may prove to be insensitive to penicillin.

Other indications are to relieve symptoms and to support the heart. It is debatable whether digitalis should be given as a routine. Recently it has been the practice not to prescribe digitalis unless there is reason to anticipate trouble with the heart. In most cases the patient can contend with the disease without support and it is better not to introduce a possible gastric irritant. Where there is extensive involvement of the lung, where there has been previous heart trouble or a history of alcoholism, it may be wise to take the precaution of giving digitalis at once. The cough may be controlled by a simple linctus, such as Gee's linctus, but in severe cases it may be desirable to give heroin. Should insomnia be a prominent feature, it is necessary to give morphia in  $\frac{1}{4}$ -grain doses, and these may be repeated nightly for so long as necessary. Paraldehyde in doses of two or three drachms is very useful in patients who do not tolerate morphia. In alcoholics it is as well to give brandy from the start of the illness, about 6 ounces daily. Should acute cardiac weakness develop, coramine, caffeine, and strychnine may be given subcutaneously and, in urgent cases, atrophanthin intravenously. Certain special indications may occur during the course of the illness. When there is much sweating, or high fever, tepid sponging gives great relief. Oxygen is rarely necessary except in severe cases. The indication used to be cyanosis, but this is not a reliable guide when



sulphonamides are being given. It is usually possible to judge, from the general condition, whether oxygen is likely to be beneficial. It is best administered through a B.L.B. mask.

When there is much congestion and cyanosis, evidence of dilatation of the right heart, venesection is indicated. As much blood as possible should be removed, and it must be remembered that, owing to the viscosity of the blood, it is often not possible to remove more than 10 ounces. In cases of difficulty it is necessary to open the vein by the old-fashioned method, using a bistoury. Local measures for the relief of pain are often necessary. The "pneumonia jacket" is quite useless and should not be advised. Antiphlogistine is very comforting and, in most cases, effective. In more severe cases blisters or leeches may be applied. If the pain does not respond to these measures, it may be desirable to induce a small pneumothorax in order to separate the two layers of the pleura. A small fill of about 200 c.cs. may be given, and this may be repeated daily for two or three days in order to keep the layers of the pleura apart. Induction of a larger pneumothorax, in order to rest the affected lobe, has been advocated but the results are not encouraging and the method is not now practised.

## CHAPTER 24

### BRONCHOPNEUMONIA

**BRONCHOPNEUMONIA** tends to occur at the extremes of life and it differs from lobar pneumonia in that the infection is usually mixed, with relatively few pneumococci and a preponderance of streptococci or Pfeiffer's bacillus as a rule. The distribution is more widespread, yet the tissue resistance is much greater, and the infection is confined to lobules instead of sweeping through an entire lobe. In bronchopneumonia there is damage to the bronchial walls and consequently bronchiectasis is more likely to result.

*Pathology.*—Both lungs are usually affected. There is always acute inflammation of the bronchioles, many of which are plugged with muco-pus. Patches of collapse and of acute emphysema are commonly present in addition. The appearance of the lungs is therefore one of scattered nodular consolidation surrounding bronchioles. In one type the infection may be confined to one lobe and may be so extensive as closely to resemble lobar pneumonia. It is sometimes difficult to distinguish consolidated areas from congested or collapsed areas. The solid part of the lung is completely airless and sinks in water. There is usually an accompanying fibrinous pleurisy, and empyema is relatively common.

There is a very acute form in which bronchitis predominates. This is most commonly the case when the disease has been so acute that death has occurred within the first day or two. It is seen in epidemic influenza and in some of the rare conditions, such as *psittacosis*. On inspection of the lung there may be no evidence of gross consolidation, but under the microscope some of the alveoli are found to contain exudate. Congestion and oedema are much more prominent features and the bronchi contain muco-pus.

*Disseminated bronchopneumonia* is the common type. The lungs appear smaller than normal, but still crepitate. Nodules can be felt in their substance. The pleura may show depressed dark areas of collapse, normal areas, and raised purplish areas of consolidation. Over these the pleura is lustreless and there

may be fibrin formation. On section, the surface is dark red and irregular, with fairly well-defined areas of consolidation scattered throughout the substance, and patches of collapse and congestion. In fatal cases there is generally marked oedema as well. The solid areas vary in size and are red or yellow in colour. A small bronchiole is often seen in the centre of the lesion. Under the microscope the bronchiole is seen to be full of leucocytes and desquamated epithelial cells, and the mucous membrane is swollen and infiltrated with white cells. The alveoli are occupied by leucocytes and alveolar cells; fibrin is less abundant than in lobar pneumonia. The walls and inter-alveolar spaces are infiltrated with leucocytes and the capillaries are dilated in the affected area. The changes are most marked in the region of the affected bronchiole, and gradually lessen towards the periphery. There is no sharp distinction, as there is in lobar pneumonia, between alveoli full of fibrin and alveoli full of white cells. In the massive form the naked-eye appearance may closely resemble lobar pneumonia, but under the microscope the alveolar exudate does not conform with the lobar type; there is a definite relationship between the bronchioles and the solid areas, and fibrin is scanty. In the type which results from aspiration of septic material there is a tendency to breaking down of the normal lung tissue, so that the alveolar septa may disappear and small abscesses may be seen. There are several distinct clinical varieties of bronchopneumonia.

(a) *Primary bronchopneumonia in children.*—This occurs chiefly between the ages of 2 and 4 years and closely resembles lobar pneumonia. It often ends by crisis.

(b) *As a complication of bronchitis.*—Usually follows influenza, whooping-cough, measles, and typhoid fever.

(c) *In any debilitating condition.*—With rickets and summer diarrhoea in infants. Complicating nephritis, heart disease, and malignant disease in adults.

(d) *Complicating certain lung diseases.*—Tubercle, bronchiectasis, lung abscess, and growth.

(e) *Aspiration pneumonia.*—This may follow operations done under general anaesthesia or operations upon the nose and throat which are performed with a local anaesthetic. It may also result when a fistula occurs between the trachea or main bronchus and the oesophagus.

*The clinical picture.*—The course of the illness is much less definite than is that of lobar pneumonia. The temperature is less regular, the duration varies a good deal, and there is no crisis.

When the condition complicates bronchitis or debilitating disease it may develop gradually without any sudden change in the symptoms or signs which would indicate the lung involvement. There is a gradual increase in the symptoms already present, and a rising temperature, pulse, and *respiration rate*. It is the respiration rate which rises most sharply and which shows the commencement of the change. In other cases, following aspiration of infected material, and in epidemics of influenza, the onset may be abrupt, with a rigor, high fever, paroxysms of irritative cough, and even haemoptysis. When the condition is well established it is found that there is irregular pyrexia, reaching from  $102^{\circ}$  to  $105^{\circ}$ , with marked variation. A very high temperature, and a slightly raised temperature with signs of extensive consolidation, are both bad signs. Cough is usually a prominent feature and it is commonly productive, although the proportion of sputum to cough varies greatly. The sputum may be mucopurulent or blood-stained. Dyspnoea is in proportion to the extent of the lung involvement. The respiration rate is always considerably raised and it gives some guide to prognosis. In children there is a reversal of the normal respiratory rhythm which is to some extent diagnostic.

On examination the degree of prostration varies greatly in different patients according to the extent of the lung damage, the virulence of the infection, and the resistance of the patient. The general appearance forms perhaps the most reliable guide in prognosis. Cyanosis is present only when there is extensive disease. The degree of dyspnoea is of some significance, and it may be found that the accessory muscles of respiration are in action, there may be retraction of the intercostal spaces during inspiration and the alae nasi may be working. There are no signs of localized disease on inspection or on palpation; the mediastinum is not moved. On percussion there may be no abnormal findings unless the consolidation is massive; with small areas of consolidation, especially when there is much surrounding emphysema, the note may be normal. The breath sounds vary, but they are usually weak in places, and bronchial breathing may be heard. The most characteristic finding is

the presence of fine or medium rales which have a metallic quality.

The prognosis is more difficult to assess than in lobar pneumonia, for there is *no set clinical course and the disease* may wander or spread in spite of efforts to stop it. Extensive bronchopneumonia causes severe toxæmia with consequent dilatation and failure of the right heart, which is the common cause of death. It is impossible to state the average mortality, for it depends largely upon the cause of the condition; the severity of the epidemic form varies greatly from season to season.

When recovery occurs there is gradual resolution and absorption of the inflammatory products. The process is not so definite as it is in lobar pneumonia, and fibrosis of the lung and dilatation of the bronchi are fairly common sequels. Sometimes the lung breaks down and forms an abscess. A pre-existing tuberculous focus may be activated and the condition may merge into pulmonary tuberculosis.

The pleura is commonly involved and pleural effusion occurs frequently. The fluid may be clear or turbid, and empyema is likely to develop. Bilateral empyema is more common in bronchopneumonia than in lobar pneumonia.

### *Differential Diagnosis*

*Acute bronchitis.*—It is sometimes difficult to be sure whether the condition is an acute generalized bronchiolitis or a bronchopneumonia. The conditions are similar in pathology, and tend to merge so that, from the point of view of treatment, the distinction is not important, although the prognosis is much worse with bronchopneumonia. The respiration rate is a useful guide, especially in children, and a rise above 40 per minute nearly always indicates consolidation.

*Lobar pneumonia.*—Massive bronchopneumonia which is confined to one lobe may closely simulate lobar pneumonia. The treatment and prognosis are much the same and therefore the distinction is not of great importance. Clinically it is often impossible.

*Tuberculous bronchopneumonia.*—Tubercle should always be suspected when the temperature persists for more than two weeks. In any case it is always a wise precaution to have the sputum examined for tubercle bacilli. Wasting and night sweats are suggestive of tubercle. An X-ray of the chest may show apical cavitation, or old calcification, but the appearances of consolidation on the film are identical in tuberculous and in simple bronchopneumonia.

*Multiple lung abscesses.*—These are a result of aspiration bronchopneumonia and therefore the diagnosis is of no practical importance.

*Multiple septic infarcts.*—This condition is part of a generalized pyaemia. The clinical picture is that of the primary condition, and the lung symptoms and signs are usually slight. The prognosis is so bad that diagnosis is unimportant.

*Treatment.*—The course, prognosis, and treatment of the disease are now to a very large extent determined by the result of a bacteriological examination of the sputum carried out at the earliest possible moment. As the infection is usually found to be mixed, it is quite probable that there will be an indication to give penicillin and a sulphonamide simultaneously. Some of the organisms may be feebly sensitive to penicillin, and in such cases it is justifiable to try the effect of larger doses than would be given in the ordinary way (p 78).

It is comparatively uncommon to find that the majority of the organisms fail to respond to penicillin and sulphonamides, although this may occur when *B. Friedlander* and *H. influenzae* predominate. In these cases the treatment consists in careful nursing, plenty of fluids, and cardiac stimulants when required; the illness may be prolonged and the prognosis is much less favourable.

There is some tendency to relapse during convalescence and therefore great care must be taken. The condition is a debilitating one, and the patient should not be allowed to return to normal surroundings until it is clear that recovery of health is quite complete.

### *Special Forms of Bronchopneumonia*

(a) *Influenza.*—Influenza is an acute generalized infection which seems, in many cases, to manifest itself chiefly in the respiratory tract. It occurs in pandemic form occasionally, the last big outbreak being in 1918. It also occurs in epidemic form in the winter months. It is characterized by variability, both in the severity of the symptoms and in the system chiefly affected. Sometimes the chief manifestations are respiratory; at other times they are gastro-intestinal, while in other seasons the symptoms may be those of an acute toxæmia. The condition is due to infection with a virus; various organisms, including Pfeiffer's bacillus, are commonly found as secondary invaders.

## 192 DISEASES OF THE RESPIRATORY TRACT

The incubation period is short, being from one to three days as a rule.

The onset of symptoms is sudden, with severe *headache*, *pains in the back*, and marked *prostration*. There is fever but the pulse rate is often relatively slow. *Coryza* is common and some degree of bronchitis is usual. In uncomplicated cases the fever lasts for three or four days and then resolves, but there is a tendency to relapse. When the respiratory tract is seriously involved there is invariably a bronchopneumonia. Post-mortem examination shows intense congestion of the trachea and main bronchi, and the consolidated areas are frequently *haemorrhagic*.

The clinical course of the bronchopneumonia is the same as that already described, but the *toxaemia* is usually more marked and, in severe cases, there may be a typical "*heliotrope cyanosis*"; the prognosis in these cases is bad.

Complications are very apt to occur. The *toxaemia* affects the heart muscle and may lead to oedema of the lungs and death from heart-failure. *Bronchiectasis* is a common sequel and it may appear close upon the illness. *Empyema* is more common with influenzal bronchopneumonia than with the other types, and it is usually streptococcal. There is often great mental depression during convalescence.

The diagnosis is easy during an epidemic. It is common to regard most febrile disorders which occur in the winter as "*influenza*", and the term is probably used to include a variety of respiratory infections.

The patient must be kept in bed until the temperature has been normal for at least a week, and until the sedimentation rate is nearly normal. Aspirin, preferably combined with Dover's powder, is effective in relieving the general symptoms. The treatment of the respiratory complications follows the lines described for pneumonia. It is not yet possible to assess the value of chemotherapy in *influenzal pneumonia* but it is greatly to be hoped that our present drugs will prove adequate to control an epidemic if such should occur. Quinine has a great reputation, not entirely undeserved, in the treatment of *influenza*.

A careful convalescence is always necessary, partly because there is a risk of relapse and partly because there is so often a well-marked *neurasthenia* from which recovery is likely to be slow.

(b) *Typhoid fever* and the paratyphoid infections sometimes cause bronchitis or bronchopneumonia, and occasionally the picture of an acute respiratory infection may predominate. In such cases the true cause may escape recognition, but severe headache, delirium, and insomnia are always suggestive features. The Widal reaction clinches the diagnosis when the physician happens to think of having it done.

(c) *Pneumonic plague*.—This is due to infection with *B. pestis* and can be spread by direct contact, for the sputum contains the organisms in large numbers. The onset is sudden, with extreme prostration, and the disease is nearly always fatal. The occurrence of several cases of severe bronchopneumonia in a family should raise a suspicion of plague.

(d) *Psittacosis*.—This is caused by a filterable virus and is conveyed by parrots and budgerigars; it does not seem to be transmitted from man to man. There is a sudden onset of symptoms with high fever, and the patient rapidly sinks into a typhoid state. There is commonly stupor and delirium. Pulmonary symptoms and signs develop towards the end of the first week with spasmodic cough, scanty sputum, and signs of patchy consolidation or of diffuse congestion. The mortality is about 33 per cent. In fatal cases it is usual to find a peculiar loose haemorrhagic type of consolidation.

(e) *Anthrax (wool-sorters' disease)*.—A severe haemorrhagic form of bronchopneumonia results from inhalation of the anthrax bacillus. The prognosis is bad.

(f) *Tularaemia*.—This occurs chiefly in the United States. An unusually severe bronchopneumonia in a patient who has been engaged in handling rabbit skins should suggest the possibility of the condition and confirmation can be obtained by finding that *B. Tularensis* is agglutinated.

(g) *Lipoid pneumonia*.—Most types of oil are toxic to the lung tissue, especially those of animal origin. Cod-liver oil may find its way into the lungs of children, and liquid paraffin, used to lubricate the nasal passages, must often pass the larynx in adults. The irritation sets up a subacute or chronic inflammation in the lower lobes and may cause diagnostic difficulty. Examination of the sputum under the microscope will reveal the presence of intracellular and extracellular globules of oil.

### Pneumonitis

#### (Primary Atypical Pneumonia)

This term is employed to describe a benign inflammatory consolidation of the lung. It is extremely common, either as a primary event or as a complication of some other infection of the respiratory tract; in fact, X-rays have shown that patches of consolidation of this type are frequently demonstrable during



## 196 DISEASES OF THE RESPIRATORY TRACT

The following table serves to illustrate the relations between the various acute lung infections :—

| <i>Resistance to Infection</i> |       | Result       |
|--------------------------------|-------|--------------|
| General                        | Local |              |
| Good                           | Good  | Pneumonitis  |
| Good                           | Bad   | Lung Abscess |
| Bad                            | Good  | Pneumonia    |
| Bad                            | Bad   | Septicaemia  |

## CHAPTER 25

### LUNG ABSCESS

Lung abscess may be defined as "non-tuberculous suppuration, with cavitation, occurring in the lung tissue". This definition makes it clear that lung abscess is a lesion of the lung parenchyma itself, and it avoids the confusion which sometimes arises between a true lung abscess and a large bronchiectatic cavity. The term "gangrene" is better avoided in the case of those lesions which are primarily infective in nature, the only possible significance of this term being that it might indicate a special type of abscess which results from anaerobic infection. Gangrene in other parts of the body results from vascular obstruction as a rule, and the term might be more suitably employed for those rare cases in which there is necrosis of a portion of the lung as a result of vascular obstruction; in order to avoid any possible confusion, however, it is better to employ the term "massive necrosis" for these cases and to abandon the use of the term "gangrene", so far as the lungs are concerned.

*Ætiology.*—The conditions which commonly give rise to pulmonary suppuration may be summarized as follows:—

#### 1. *Single Lung Abscess.*

##### (a) *Lesions of the respiratory tract.*

(i) *Mouth and pharynx.* Tonsillectomy, dental extraction.

(ii) *Larynx and trachea.* Post-operative.

(iii) *Bronchi.* Foreign body, carcinoma, perforation from oesophagus, rarely with bronchiectasis.

(iv) *Lungs.* Pneumonia, pneumonitis, actinomycosis and fungus infections, rarely chest injury.

(b) *Abdominal condition.* As a complication of abdominal operations, especially in a septic field.

(c) *Peripheral septic conditions.* Acute infections of bones, joints, and skin.

(d) *Primary group.*

#### 2. *Multiple Lung Abscesses.*

(a) In pyæmia.

(b) Following the aspiration of septic material.

(c) In fatal cases of bronchopneumonia.

The causation of lung abscess has been the subject of much discussion. There are two chief theories, the aspiratory and the embolic. It might be thought that the aspiration of septic material into the bronchial tubes would inevitably lead to suppuration, but in practice it is found that, *when the cough reflex is active*, the foreign matter is usually expelled without giving rise to trouble. Animal experiments have confirmed this view, and it has been shown that lung abscess is much more likely to develop when, in addition to aspiration, there is some cause of retention of aspirated material in the bronchi. The additional factors are *depression of the cough reflex*, which may result from the administration of morphia before or after the anaesthetic, and bronchial obstruction, which is often due to excessive drying up of the mucus in the tubes when atropine has been given pre-operatively.

The impaction of a septic embolus in a small branch of the pulmonary artery is an obvious possible cause of lung abscess. The lung abscess which complicates a peripheral infection, such as a whitlow or a carbuncle, is clearly embolic. Embolic abscesses are frequently multiple, but they may be single.

It is probable that cases in which lung abscess follows operations on the tonsils and upper respiratory tract are usually aspiratory, but it must be remembered that a local pulmonary factor, such as collapse, must also be present. The cases which follow abdominal operations may be either embolic or aspiratory, but probably the former as a rule. Lung abscess does not appear to follow clean abdominal operations, and therefore it is not easy to see how aspiration can play an important part. In this group the lesions are commonly situated in the right lower lobe close to the diaphragm, and it is possible that some of these result from a direct lymphatic spread.

An abscess is essentially a peripheral lesion and it is always close to the pleura. An apparently deep-seated abscess lies adjacent to the interlobar pleura and may closely simulate an interlobar empyema (figs. 23, 24).

Lung abscess may be unilocular or multilocular and it may be "open", that is, in communication with the bronchus, or "closed". It may also perforate the pleura, forming a broncho-pleural fistula.

The common causal organisms are fusiform bacilli and

spirilla, pneumococci, staphylococci, and streptococci. Foul pus is always the result of fuso-spirochaetal infection.

The clinical picture of lung abscess varies a great deal in different cases.

(a) In patients who have recently had pneumonia, and in children, especially following tonsillectomy, the symptoms may be those of a mild general toxæmia with slight fever; there may be no respiratory symptoms nor signs whatever, and in such cases the diagnosis must depend upon the X-ray examination of the chest (fig. 25).

(b) In other cases there may be a much more striking general reaction with high fever, and in these there are usually definite respiratory symptoms and signs. *Cough* occurs early, and is often paroxysmal at first. In this stage there may be no sputum, or perhaps a little blood. When an abscess ruptures into a bronchus there may be a considerable quantity of pus which is by no means always offensive. In very acute cases the condition may be indistinguishable from bronchopneumonia. It must be remembered that this type of condition may be a primary event or it may follow some other condition; in either case copious purulent sputum, of sudden onset, should suggest the possibility of abscess.

(c) In the common type the picture is that of a subacute or chronic respiratory disease, with cough, sputum, and evidence of general toxæmia. The symptoms depend to some extent upon the exact situation of the abscess in relation to the surrounding structures. When the lesion is situated near a main bronchus, *irritating cough* and *haemoptysis* are frequent symptoms. At first the cough may be unproductive, but later the abscess is likely to rupture into a bronchus, and the sputum is copious and purulent; the *odour* varies according to the nature of the infecting organisms. Should the infection be due to fusiform bacilli and spirilla, as is often the case, the sputum will be typically foul-smelling, and this same odour is often noted in the breath, even before the abscess opens into a bronchus. With pneumococcal, streptococcal, and staphylococcal infections there may be no odour whatever. It is customary to regard foul sputum as being characteristic of lung abscess, but this is true of only one type, and the absence of odour in the sputum does not by any means exclude lung abscess. When the abscess is situated under the visceral pleura

there is usually acute pleurisy and the patient frequently complains of *pain* on breathing. The presence of pain should suggest that the lesion is superficial.

As has already been stated, the abscess may resolve spontaneously within a few weeks, but in some cases it becomes chronic and then there are persistent cough and sputum, so that the clinical picture may come to resemble the suppurative type of bronchiectasis, which may, indeed, supervene.

Physical examination of the chest yields very divergent results. In some cases, when the abscess is situated deeply in the lung tissue, there may be no physical signs of any sort. In others, situated more superficially, or larger in size, there may be restriction of movement on the affected side, impairment of percussion note, bronchial breathing, and rales. Pleural friction is sometimes heard. Occasionally the signs of pleural effusion may mask an underlying abscess. Clubbing of the fingers is common. A symptom suggestive of lung abscess is that cough and sputum may be produced by sudden *change of position*, and the physical signs are found to alter considerably after a quantity of pus has been coughed up.

*Complications.*—Severe haemoptysis is rare, but it may prove fatal. Acute pleurisy is common, and an *empyema* may result. The rupture of the abscess into the pleura may produce a *pyopneumothorax*. A serious consequence of chronic pulmonary suppuration is *cerebral abscess*.

The exact diagnosis must depend upon a consideration of the symptoms and signs, together with the results of special investigations.

1. *The sputum.*—The physical characters of the sputum have already been described and have been shown to vary a great deal but, when the abscess has ruptured into a bronchus, the sputum always consists of pus. Tubercle bacilli should always be sought, but they should not be found in the cases of simple lung abscess. They may of course be present if an abscess has lit up a quiescent tuberculous focus. Numerous other bacteria may be present, of which the most striking are fusiform bacilli and spirilla. These organisms are not constant, and they are also found in the sputum from cases of bronchiectasis and bronchial carcinoma, so that they are not themselves of great diagnostic value. The most significant finding in the sputum is the presence of elastic fibres, which are indicative of a lesion in the lung tissue, and which therefore form a useful point of distinction between lung abscess and bronchiectasis.

2. *Radiology*.—An X-ray should always be taken in cases in which there are symptoms or signs of respiratory disease, or pyrexia of uncertain origin. It is essential whenever a lesion is demonstrated on the film, to take a lateral as well as an antero-posterior view. There are certain characteristics of lung abscess which are fairly constant. The shadow is usually circular and fairly well defined (fig. 25). The most definite diagnostic point is the presence of a fluid level in the substance of the shadow itself (fig. 23). This occurs only when there is a cavity which contains both fluid and air, and lung abscess is the commonest cause of this combination in the lung. A fluid level may be seen when there is air and fluid in the pleural cavity, and it is sometimes difficult to be certain whether the suppuration is in the superficial part of the lung or in the pleura. In such cases it is useful to remember that the fluid level in lung abscess never extends so far as the rib margins, whereas, with fluid in the pleura, the level always extends out to the chest wall. In some cases of interlobar empyema the differential diagnosis may be very difficult, especially when the empyema has ruptured into the lung.

A bronchogram helps to distinguish lung abscess from a bronchiectatic cyst. Lipiodol will not usually enter a lung abscess cavity, being prevented by swelling of the mucous membrane of the bronchus leading to the affected area (fig. 18), whereas it will enter most bronchiectatic sacs (figs. 15, 16).

Sometimes the abscess is not visible because of the density of the surrounding inflammation. A tomogram is very useful in demonstrating the presence of cavitation in such cases.

3. A routine *bronchoscopy* should be done in all cases in order to exclude the possibility of a foreign body or of neoplasm. Either of these may be present without any characteristic history being obtained, and it is important that the possibility of their presence should be excluded. Of course, when a foreign body is present, it is removed through the bronchoscope if possible.

4. Exploratory needling of the chest is very rarely necessary in order to determine the situation of the pus. When the pus is in the lung and the pleural cavity is free, there is a danger that this procedure may cause a pyopneumothorax. It is always wiser not to explore the chest with a needle in cases of suspected lung abscess.

*Treatment*.—The management of a case of lung abscess demands great subtlety, for many variable factors are concerned and a great diversity of methods are required to meet the different conditions which may be present. In the first place it must be acknowledged that a considerable proportion of all cases of lung abscess, no one can say how many, recover spontaneously after the rupture of the abscess into a bronchus (fig. 25). The corollary of this is that there is hardly ever any

indication for urgent operation in a case of lung abscess and, in practice, the consideration of surgical drainage should be postponed for six or eight weeks.

The chief factors which influence the decisions which have to be made are the nature of the infecting organisms, the cause of the abscess, the site and extent of the lesion, the general condition of the patient, and the presence of complications.

The prevention of lung abscess is a matter of considerable importance. Chemotherapy may be used prophylactically in order to guard against infective complications when operations on the upper respiratory tract, or elsewhere, are thought to entail a risk of pulmonary suppuration. When the physical signs in the chest suggest that a part of the lung is about to become deflated, the administration of carbon dioxide may help to keep it expanded. When there is evidence of actual bronchial blockage, by mucus or muco-pus, immediate bronchoscopy may free the passage and so minimize the likelihood of an abscess. Proper ventilation of the whole of the respiratory tract would appear to be the best insurance against the occurrence of pulmonary suppuration.

Multiple lung abscesses are usually pyaemic in origin and the outlook in such cases is not good. Nevertheless the prompt exhibition of sulphonamide or penicillin in sufficient dose holds out a fair prospect of recovery. The technique of administration of these drugs is dealt with elsewhere (pp. 78, 183).

The treatment of single abscess is much more important, for, with proper management, the results should be good.

So long as the patient shows signs of good resistance to the infection, it is desirable that medical measures only should be adopted for a period of at least two months. Medical treatment consists of rest in bed, postural treatment, and drugs. Postural treatment has gained greatly in popularity in the last few years, and it has enabled many patients to avoid operation. The posture adopted must depend entirely upon the situation of the abscess and, with cavities situated near the base of the lung, it is essential that adequate drainage should be secured by placing the patient upon a properly constructed posture bed, on which the correct position for drainage may be maintained for many hours at a time. A record of the quantity of sputum should be kept daily, and it is not difficult to predict the probable effect of the treatment within a short while of its commencement.

The appropriate drug treatment should always be instituted without delay. It is nowadays vital that the bacteriology of the sputum should be investigated at once, and penicillin, sulphonamide, or both together, should be given in full doses according to the indications afforded by this examination. Even when only a proportion of the organisms are found to be sensitive, it is advisable to give penicillin in the hope that the resistance mechanism of the patient may be able adequately to deal with those which are left.

Although an initial bronchoscopy is desirable, as has already been stated (p. 201), repeated bronchoscopy with a view to washing out the abscess cavity is not of much value. This method of treatment does not appear to possess any advantages over postural drainage, and it is much more troublesome to carry out. The induction of a pneumothorax is sometimes advised. This should never be attempted when the abscess is situated near the visceral pleura, as there would be great risk of causing an empyema if this were done. When the abscess is deeply situated, and is draining freely into the bronchus but is not showing signs of healing, an artificial pneumothorax may be induced in the hope of obliterating the cavity. Physical methods of treatment, such as the local application of heat, have been used since the time of Hippocrates. Very recently it has been claimed that it is possible to apply heat directly to the deep tissues by means of ultra short wave diathermy, and this method has therefore been strongly advocated in pulmonary suppuration. The results should for the present be accepted with some reserve, in view of the fact that a fair percentage of cases heal spontaneously.

In cases which are becoming chronic it may be necessary to advise surgical treatment. By far the most satisfactory operation is a simple drainage of the cavity. This should be done in two stages. At the first operation a rib resection is performed, and the layers of the pleura are stimulated to adhere. Ten days later, when the adhesions should have become sufficiently firm, the abscess is opened and a drainage tube is inserted. In cases where the pleura is found to be already adherent at the time of the first operation, the abscess may be opened without waiting. Most cases do well following drainage by this method, but occasionally a broncho-pleural fistula may result or, for some other reason, the cavity may



persist. In such cases it is essential to do something more, and the affected part of the lung may be excised with a cautery, or a lobectomy may be performed.

When an empyema is already present the condition should be treated as such. With streptococcal infections an intercostal drainage tube may be inserted and negative pressure drainage carried out, although further operation is likely to be necessary. When the pus is thicker, as occurs with pneumococcal or staphylococcal infections, rib resection and open drainage are best. The condition usually heals quite quickly but, if a broncho-pleural fistula remains, chronic empyema is likely to result.

## CHAPTER 26

### PULMONARY TUBERCULOSIS

TUBERCULOSIS is a very common disease which results from infection with the tubercle bacillus. The exact study of the pathology dates from 1882, in which year Koch discovered the causal organism.

*Routes of infection.*—Tubercle bacilli have not been found extensively distributed under natural conditions. They are commonly present in unpasteurized milk, in "droplets" coughed up by an infected person, and sometimes in dust. They may therefore be ingested with contaminated food or they may be inhaled by those in contact with an individual suffering from the disease. The bacilli may reach the lung either by way of the respiratory passages or by the blood stream. *The balance of probability strongly favours the air-borne route as the cause of respiratory infections.* The fact that the majority of cases of pulmonary tuberculosis are due to the human bacillus, whereas most cases of primary abdominal tuberculosis result from bovine infection, is difficult to explain in any other way.

*Microscopic features.*—The early stages of tuberculosis of the lung are best studied under the microscope. The organism gains entrance through the mucous membrane of the bronchus, often without leaving any trace of its passage. It may there be taken up by a polymorphonuclear cell and carried to the lymphatics. Sometimes the cell kills the bacillus, but frequently the cell itself is killed and it is then ingested, with the living bacillus, by a mononuclear cell which conveys it to the neighbouring lymphoid tissue where it comes to rest and multiplies. *The early lesion is therefore in the peribronchial lymphoid tissue.*

During the first week after infection there is a steady proliferation of large endothelial cells ("epithelioid" cells). These are definitely not epithelial, and it is most probable that they are derived from the histiocytes which form part of the reticulo-endothelial system; it is more likely that they are derived from the fixed cells of the tissues than from wandering cells in the blood. The developing tubercle is composed chiefly of these cells. In due course "giant cells" appear which are much larger than the other cells in the lesion; they are round and contain numerous nuclei situated at the periphery. It is considered that they are formed by the fusion of several endothelial

cells and that they digest tubercle bacilli.

After a few days the developing tubercle is surrounded by lymphocytes derived from the neighbouring lymphoid tissue, and the typical appearance of tuberculosis is then complete. A very characteristic feature is the lack of blood vessels, which is responsible for the tendency to necrosis to which the term *caseation* is applied. In the central part of the lesion the cells degenerate, the nuclei disappear, and eventually all structure is lost. There is a tendency for the necrotic process to extend outwards. It is thought to be caused partly by lack of blood supply and partly by the local action of the toxin derived from the bacilli. In cases where the infection is very mild, and where the tissue reaction is sufficient, there may be complete absorption of this early lesion. In many cases there is a fibrous tissue reaction around the tubercle which encloses it in a capsule, and in this state the lesion may remain quiescent for a long period. The final stage in healing is the deposition of calcium salts.

The tubercle which has just been described is microscopic, but, in the course of time, multiple tubercles develop and a macroscopic, seed-like tubercle is produced, the *grey tubercle*. As the infection extends, and the tubercles coalesce, the area of caseation also becomes visible, and the later stage, the *yellow tubercle*, is present.

Miliary tubercles always indicate recent and active disease, at which stage endothelial cells are prominent. Giant cells and caseation indicate that the bacilli have overcome the local resistance, whereas plentiful lymphocytes and active fibroblastic reaction show an attempt to repair the damage. The extent of the fibrosis is a measure of the resistance, but fibrosis leads to quiescence rather than to arrest. The soundest form of healing is that associated with calcification, although living bacilli may be present even in the calcified lesion. The blood vessels in the affected area show endarteritis, with marked thickening of the intima. This change leads to thrombosis and it is therefore a safeguard against severe haemorrhage. There may also be changes due to secondary infection.

The changes in the surrounding tissues are partly toxic and partly mechanical; many of the alveoli are compressed or collapsed and others may contain plasma and catarrhal cells.

The following is a summary of the changes which may be found under different circumstances. When resistance is very low the necrotic process is much in evidence, tubercles are numerous and giant cells are present, but there is little or no fibrosis. In extreme cases there may even be no giant cells nor tubercle formation. When the resistance is better the classical picture is that of a mixture of fibrosis and caseation. With very good resistance there may be much formation of fibrous tissue and little or nothing to indicate the tuberculous basis of the condition.

*Morbid anatomy.*—The four chief changes in the lungs are

*caseation, cavitation, fibrosis, and calcification.* The initial lesion is *peribronchial* and consists of an aggregation of grey tubercles which fuse and form one or more yellow tubercles. The subsequent course is determined chiefly by the *resistance of the patient*, which varies from time to time. Adequate defence results in *fibrosis* and the development of scar tissue, often with some *deposit of calcium*. An advancing lesion is shown by increase in the size of the caseous area with little or no fibrosis. In the course of time the lesion breaks down and its contents are discharged into the bronchus. This forms the commencement of the *cavity*, the stigma of active tuberculosis in the lung. It is held by many that secondary infection is a common precursor of cavitation. The further course depends upon the ability of the tissue to put up a defensive reaction, by means of fibrosis, even at this late stage. The recent active cavity has soft, ragged walls, whereas the chronic, rather quiescent, cavity has smooth shiny walls, shut off from the lung by a layer of fibrous tissue. It is a characteristic feature that the lung tissue may be completely destroyed while the bronchi and the blood vessels are little affected. In time the vessels are weakened and small aneurysms may form and rupture, but as a rule thrombosis occurs first and the vessel is effectively sealed.

In patients who have chronic active tuberculosis the fibrous tissue formation may not be sufficient entirely to obliterate the lesion and tubercles may be seen at the periphery. It may therefore happen that the disease is quiescent in some parts while it is actively extending in others.

*Calcification* is the soundest form of healing, apart from complete absorption of a lesion, and the process can be followed on serial X-rays. In the course of years a "lung-stone" may be found, and this may cause haemoptysis purely from mechanical injury to the weaker blood vessels. But the disease is sometimes seen to be active in some directions while it is calcifying in others, so that the films must be studied carefully before any opinion is expressed that the disease is fully quiescent.

In nearly every case the overlying pleura is involved by fibrinous pleurisy at first, which later develops into an area of thickening and adhesion to the chest wall. This adhesion is of the utmost importance, for it may seriously hinder the collapse of the diseased part of the lung during pneumothorax treatment.

Tubercle bacilli often gain entrance to the blood stream in small numbers. The condition is a bacillaemia and it may give rise to metastases in the bones, joints, kidneys, or the nervous system. When a sudden and extensive invasion of the blood stream takes place, as occurs when the contents of a caseous gland are discharged into a blood vessel, the resistance is overwhelmed and *acute miliary tuberculosis* results. Invasion of the pulmonary artery causes the lung to be chiefly affected, but, if the material gains entry to the

pulmonary vein, the result is a generalized miliary tuberculosis. This condition is nearly always fatal, but occasionally a patient is found who has sufficient resistance to convert the condition to a chronic state, and recovery has been known to occur when the lungs have been chiefly affected.

*Ætiology.*—Dwellers in cities, subject to repeated sub-infection, develop considerable immunity, although this may be overcome, in the individual, by unfavourable circumstances. Country dwellers come less into contact with the infection and are therefore less immune. Native races, previously unexposed to infection, fall ready victims when brought into contact with civilization, and develop massive lesions. The mortality in this type of case is very high.

If we accept the fact that most of us are repeatedly infected with tuberculosis in the course of our lives, it follows that the resistance of the majority to the infection must be very great. Clinical tuberculosis results only when an individual of average resisting power meets with an overwhelming infection, or when average resistance is temporarily lowered and a comparatively minor infection is enabled to gain a foothold. Hence the very great importance of the maintenance of general health on the part of the individual, and the necessity for the avoidance of those factors which are well known to lower it. The margin between health and tuberculosis is not great and anyone may develop the infection in suitable circumstances.

A most important question concerns the infectivity of the condition. Wingfield has pointed out that pulmonary tuberculosis is not transmitted *as such*, but that it is the generic disease *tuberculosis* which is transmitted. The development of pulmonary tuberculosis in adults depends upon many factors, such as sensitiveness resulting from a previous primary infection, the general health of the individual, the power of resistance, and the virulence and dose of the infecting organism.

Constant contact with infection is a most important factor in the transmission of the disease, especially in the case of children of tuberculous parents, and there is evidence that there is a significantly higher incidence in married couples when one is known to have been infected previously.

*Heredity.*—It is certain that pulmonary tuberculosis is not inherited *as such*. It is true that more than one case often occurs in a family, but contact with the infection and bad

surroundings account for most of these cases. There are, however, certain families in which there appears to be an hereditary susceptibility to the disease.

*Age incidence.*—The common primary infection of young children does not produce symptoms and is therefore usually overlooked. Clinical tuberculosis in children is almost always acute, either miliary or pneumonic, and usually fatal. Chronic pulmonary tuberculosis is uncommon before puberty.

The immunity acquired in childhood may wear off during adolescence, and pulmonary tuberculosis becomes common between the ages of 16 and 30 years. This type is often rapidly progressive and the outlook is not good. The incidence of chronic pulmonary tuberculosis becomes gradually less towards middle age, but there is still a considerable mortality amongst those who have acquired the infection as young adults.

*Sex.*—On the whole males are more commonly affected, but females predominate in the young adult group. This appears to be partly due to the strain of puberty and of pregnancy, both of which are considerable. Curiously enough the symptoms are often much improved during pregnancy, but they tend to return in an exaggerated form shortly after the child is born. In many cases the first symptoms of rapid consumption develop during lactation.

*Environment.*—The social status of the patient exercises a determining influence upon the development of the disease, and upon its prognosis. Overcrowding, insufficient food, poor ventilation, long hours of work, tiring journeys, and mental stress, which is very likely to be the result of financial worry, are each important. Excessive physical exercise is a contributory factor.

*Occupation.*—Apart from overcrowding and bad conditions of work, occupation is chiefly important so far as silicosis is concerned. Those whose occupation exposes them to silica dust are likely subjects for phthisis. The relationship between the two conditions is discussed in a later section.

*Associated diseases.*—A quiescent focus is apt to be activated by acute infections, notably whooping-cough, measles, influenza, and typhoid fever. Pneumonia does not appear to predispose towards tuberculosis.

In certain chronic wasting diseases, notably *diabetes*, *cirrhosis of the liver*, and chronic nephritis, tubercle is a common

complication. Alcohol tends to lower resistance and the alcoholic subject who contracts the disease rarely does well. Tuberculosis is not uncommon among patients in mental hospitals.

In *congenital heart disease* the lungs are often small and poorly developed and form a suitable soil for the spread of the infection.

It is stated that *phthisis* is unlikely to occur in those who have mitral stenosis, gout, or tuberculosis of the bones, glands, or joints. In this connection, however, it is necessary to quote Louis' law, which asserts that any tuberculous lesion in the body, occurring after puberty, is likely to be associated with pulmonary tuberculosis.

The question of tuberculosis following injury to the chest has to be considered in view of its possible legal consequences. Injury cannot *cause* tuberculosis, but it might easily activate a quiescent focus or aggravate an unrecognized patch of active disease. It must be remembered that the injury may draw attention to disease unsuspected but already active, and so it may sometimes prove actually to be to the advantage of the patient.

*Allergy in tuberculosis.*—It has been suggested that many of the phenomena of pulmonary tuberculosis are due to hypersensitiveness of the tissues to the proteins of the tubercle bacillus. This view has been expressed in particular with regard to the formation of cavities and the phenomena of epituberculosis. It is established that the occurrence of the primary complex is signalized by the development of cutaneous hypersensitivity, and this fact is of great importance in the early detection of the infection in children.

The *Mantoux test* is the most reliable index of the development of the sensitive state. The complete test should always be carried out before it is assumed that the result is negative. An injection of 0.01 mgm. of Old Tuberculin is given intradermally and the reaction is noted in 48 hours. A positive result is shown by central swelling with surrounding erythema. If no reaction is found an injection of 0.1 mgm. is given and the result is read after the same interval. A certain number of cases negative to the previous test will now be found to be positive. The negative reactors are given a final injection of 1.0 mgm. of Tuberculin. Those who show no response may be regarded as completely negative.

Recently a "patch test" has been used in order to overcome the need for injections. A small piece of gauze impregnated with Tuberculin is applied to the skin and fixed with adhesive. The results are only moderately satisfactory and are not to be compared for reliability with those obtained by intradermal injection.

The part played by allergy in determining the response of the lung to infection is not known, although much speculation has been advanced upon the subject. There is a tendency to regard allergy and immunity to tuberculosis as being closely linked phenomena but this is probably a mistake. Indeed it has been stated that there is no evidence to support the view that hypersensitiveness is necessary for the operation of immunity in any infection. The Mantoux test, therefore, is not an index of immunity or otherwise and it should not be so interpreted. It is simply an indication of infection and its chief value is in detecting recent infection.

### B.C.G. Vaccine

The *Bacillus Calmette-Guérin* ("B.C.G.") is a tubercle bacillus of attenuated virulence which has been used as a living vaccine in order to produce immunity to tuberculosis. Originally developed in France, this vaccine has recently been employed on a large scale in Norway and Sweden, and excellent results are reported. The vaccine must be freshly prepared and the best method of administration is by the "multiple puncture" technique; the vaccine is spread on the skin and a number of needle punctures is made through the emulsion. A series of small papules develops and these gradually fade, leaving little or no mark behind them.

So far as this country is concerned, there has been very little use made of B.C.G. vaccine hitherto, although it is likely that supplies will be available in the near future.

The value of B.C.G. is that it permits the immunization of groups of susceptible individuals who are likely to be exposed to infection. The vaccine must only be given to those who have not been already infected, those whose Mantoux test is negative, and it is likely to be applied chiefly in the case of children who have been living in contact with a case of open tuberculosis, nurses about to take up duty in sanatoria and



in certain general hospitals, medical students, and entrants to the Services.

The *vole bacillus* (p. 83), may also prove valuable as an immunizing agent. Preliminary results suggest that a vaccine *made from this organism is at least as effective as B.C.G. in this respect*, and it may prove that a vole bacillus vaccine is free from some of the disadvantages of B.C.G. Much further study of both vaccines will be necessary before any final conclusion can be drawn.

## CHAPTER 27

### PULMONARY TUBERCULOSIS (continued)

CLINICAL pulmonary tuberculosis may present itself in a great variety of ways and the following classification may prove helpful in indicating the chief changes which are likely to be encountered :—

#### TUBERCULOSIS OF THE LUNGS AND PLEURA

1. *The Primary Lesion.*
  - (a) Ghon's focus.
  - (b) " Epituberculosis."
2. *Acute Pulmonary Tuberculosis.*
  - (a) Bronchopneumonia (bronchogenic).
  - (b) Miliary tuberculosis (haematogenous).
3. *Chronic Pulmonary Tuberculosis.*
  - (a) Post-primary lesion (Assmann's focus).
  - (b) Caseation ("infiltration").
  - (c) Cavitation.
  - (d) Fibrosis.
  - (e) Calcification.
4. *The Pleura.*
  - (a) Dry pleurisy
    - Acute fibrinous.
    - Chronic adhesive.
  - (b) Pleurisy with effusion.
    - Fluid clear or blood-stained, empyema.
  - (c) Pneumothorax, with or without effusion.
5. *Tuberculosis of the Mediastinal Glands.*

*The primary infection.*—The careful studies of Opie have shown that *the primary infection is pulmonary* and not intestinal in most cases. In densely populated districts few children escape, but in rural areas the incidence is less. The great increase in the use of diagnostic radiology has revealed the existence of a considerable number of primary complexes in adolescents and young adults. Most of them are symptomless and heal spontaneously, so that they cannot be identified by clinical means alone.

The essential feature of the primary complex is the *Ghon's focus*. This is a symptomless lesion and is therefore unlikely to be detected in its active phase unless it is specially sought.

chest, the heart is not displaced, the percussion note is moderately impaired, and there is bronchial breathing. There may be no added sounds at first, but later, as the lung tissue breaks down, metallic rales are heard. Cavitation may result in intense (amphoric) breathing. These signs are not pathognomonic and the diagnosis hinges entirely on the discovery of tubercle bacilli in the sputum. A very suggestive sign is the failure of the temperature to respond to chemotherapy; a persistent pyrexia of more than ten days should always lead to the suspicion of a tuberculous infection.

It is occasionally impracticable to have an X-ray taken, but this must be done whenever possible in order to define the extent of the disease (fig. 37).

The general management of the acute stage is similar to that of pneumonia. Complete rest, careful nursing, proper ventilation at an even temperature of 60° F., and plenty of fluid. Symptomatic medicinal treatment consists of measures to relieve the cough, by means of a linectus, and to relieve pain, by means of local applications of heat or counter-irritants. The recognition of the condition as tuberculous implies that the prognosis is grave, and therefore additional methods should be considered at once. It is unwise to give gold during the acute stages as a rule. In suitable cases, when the disease is confined to one lung, a pneumothorax may be induced. Although this condition has a high mortality the disease may become less active in the course of time, and this process is materially assisted if a satisfactory pneumothorax can be obtained. As the patient approaches convalescence the clinical picture merges into that of chronic pulmonary tuberculosis, and treatment can be carried on along the lines laid down for the management of the chronic stage.

*Miliary tuberculosis.*—This occurs chiefly in children and in young adults. It is a rare complication of phthisis. It results from the entrance of organisms into the blood stream, usually from a hilar gland.

*Acute miliary tuberculosis* of the lungs results from the rupture of a caseous gland into a branch of the pulmonary artery or into one of the systemic veins. The clinical appearance is that of a severe infection, with high fever, tachycardia, dry cough, and *dyspnoea which is out of proportion to the signs in the lungs*. On physical examination there is little to be found

except evidence of acute emphysema and occasional metallic rales. The diagnosis is usually made on the X-ray, which presents a characteristic "snow-storm" appearance (fig. 38). There is little sputum, but the bacilli are occasionally discovered in this, in the stomach washings, or in the faeces. This condition is almost invariably fatal in a few weeks and there is no treatment.

*Chronic miliary tuberculosis of the lungs* may occur when the infection is not massive and the resistance is exceptionally good. This rare type may run a much more prolonged course and recovery is not unknown, the lesions may calcify or they may even disappear.

*Generalized miliary tuberculosis* occurs when the gland ruptures into a pulmonary vein, with dissemination throughout the body. In the *typhoidal* form the clinical picture is that of a general infection, with abdominal symptoms. The X-ray usually shows evidence of tubercle in the lungs. Diagnosis is difficult and it rests largely upon exclusion of the other causes of pyrexia. As in typhoid fever, there is often a leucopenia. *Tuberculous meningitis* is a common condition in children between the ages of 2 and 5 years. There are intracranial symptoms and signs, and the diagnosis can be made by lumbar puncture. It is almost invariably fatal. On post-mortem examination miliary tubercles are found scattered throughout the lungs as well as in other parts of the body.

### Chronic Pulmonary Tuberculosis

The mortality from pulmonary tuberculosis has been declining steadily for the last hundred years, and this is only in part due to improvement in public health control and increased efficiency in treatment. The earlier the patient comes under observation the better the chance of ultimate recovery with proper treatment, and it is therefore most important that the diagnosis should be made at the earliest possible moment. It must be conceded that a radiological lesion may be demonstrable for weeks, months, or even for years, before symptoms make their appearance. In fact, X-ray and post-mortem evidence both point conclusively to the fact that many apparently normal individuals have suffered quite severe tuberculous infection and have even overcome it, without ever having experienced any symptoms of respiratory disorder. Yet it is also true that the symptoms of early tuberculosis are either disregarded or misinterpreted in many cases, and this often happens because the earliest symptoms are apt to be those of

toxaemia (p. 22), and these often do not point directly to an origin in the lungs. *Physical signs are rarely present when the lesion is minimal, and therefore an X-ray must always be taken when there is even a remote possibility that tuberculosis may be present.*

*The post-primary lesion.*—This results when an individual, who has been previously sensitized in childhood, sustains a reinfection, usually in early adult life. It is the earliest event in chronic pulmonary tuberculosis. The lesion consists of a spherical area of consolidation 1 or 2 cms. in diameter, situated in an upper lobe an inch or two below the apex, more commonly on the right side. There may be symptoms of mild toxaemia (vide infra) or, rarely, symptoms of the local lesion, such as a small haemoptysis. Physical signs are indefinite, but crepitations may be heard. The lesion can be identified on the X-ray film, in which it appears as an *Assmann's focus* (fig. 30), a circular opacity of medium density, usually behind or below the clavicle; the hilar glands are not enlarged. This lesion may be completely absorbed in the course of a few months, it may shrink and be replaced by a fibrous scar, or it may break down and discharge its contents into a bronchus, forming a cavity. It is this tendency to cavitation which is the characteristic feature of the disease.

*Symptoms result partly from toxaemia and partly from the local disease in the lung.* The tubercle bacillus produces a toxin which acts on all the tissues of the body, causing widespread vaso-dilatation and also, to some extent, affecting the nervous and muscular tissues. Although the symptoms and signs of toxaemia and of the local lesion are usually present together, they vary greatly in their respective proportions. Thus there may be a febrile illness, with malaise and loss of weight, and yet there may be no cough nor sputum, or, on the other hand, there may be cough, with copious purulent sputum and haemoptysis, yet little fever, and the patient may feel comparatively well. An understanding of this combination of toxic and local respiratory effects is necessary in order to comprehend the very varying guises in which pulmonary tuberculosis may be encountered.

*Toxaemic symptoms.*—The patient may complain of physical fatigue, worse at

the end of the day. There is lack of muscle tone and sometimes anaemia. The mental attitude of the patient varies and, in the early stages, there may be considerable depression, but later, and particularly in the terminal stages, the mental outlook often becomes cheerful and optimistic (*spes phthisica*). *Night sweats* are comparatively common and are suggestive of tuberculosis, although they may occur with other infections and with lymphadenoma. *Dyspnoea* may be an early symptom, the cause of which is complex, but to some extent it is probably due to toxæmia depressing the myocardial function. *Dyspepsia* may be the first and, for a considerable time, the only symptom of phthisis. In the early stages it is usually of the hyperacid type and may suggest a diagnosis of gastric ulcer, or of some other digestive disorder. A patient who has unexplained dyspepsia of this type should be examined from the point of view of a possible chest lesion. In the later stages, with marked toxæmia and wasting, the dyspepsia changes to the hypoacid type, with *loss of appetite*, *nausea*, and, at times, *diarrhoea*.

*Loss of weight* is an important early symptom. It may occur in patients whose appetite is still good and it is therefore not wholly due to digestive disturbance. The rapid increase in weight which often occurs when the patient is put to rest indicates that loss of weight results chiefly from toxæmia.

*Amenorrhœa* is a not uncommon early symptom of tuberculosis in young women. It does not usually occur alone, but tubercle should always be considered when there is no obvious explanation in the pelvic organs.

Finally, the toxæmia of tuberculosis is one of the commonest causes of *obscure pyrexia*. The temperature may be sufficient to draw attention to itself or it may be so slight as only to be noticed as a result of routine observation. It is generally agreed that, provided that no liquid has been taken for at least half an hour before the observation is made, the mouth temperature is the most reliable. The rectal temperature is a little higher but it is strictly parallel to the mouth temperature and there is therefore nothing to be gained by rectal observations. Normally the variation between the morning and evening temperatures should not be more than 1° F., and an early finding in active tuberculosis is an increase in this daily variation. The most likely time to find the temperature raised is about 6 P.M., and a reading should always be taken at this time.

Physical exertion may cause an appreciable rise of temperature but this should have fallen to normal after resting for half an hour. It is probable that the temperature of uncomplicated tuberculous infection maintains a steady raised level, and this may be seen in the generalized miliary form. In chronic pulmonary tuberculosis the picture is complicated by the effects of the secondary infection which is almost invariably present, and therefore a swinging temperature, which is higher in the evening, is most commonly seen. In rare cases there is an inverse type of temperature curve, the pyrexia occurring in the morning, and it is said that this type indicates a bad prognosis. Occasionally a Pel-Ebstein syndrome may be present. The temperature chart is an excellent guide to progress and, combined with the weight record, it forms the basis of our attempt to assess the prognosis.

There is usually tachycardia, the pulse being rather soft and easily compressed. This results from the action of the toxin on the myocardium and on the blood vessels. A persistently raised *resting* pulse rate indicates a very active lesion, and a fall to normal is satisfactory evidence of diminution in the toxæmia.

*Local symptoms.*—These are the common symptoms of any respiratory infection.

*Cough and sputum* are the earliest symptoms, and perhaps the most neglected. In the early infiltrative stage there may be only a morning cough with a little mucoid or mucopurulent sputum, but, when the lesion opens into a bronchus and becomes secondarily infected, the sputum becomes frankly purulent. *Any patient who has a cough for more than three weeks, which does not respond to treatment, should have the sputum examined on several occasions.* As the lesion advances the cough becomes more pronounced as a result of the irritation of secretion stagnating in the bronchi.

In very chronic cases bronchitis and emphysema eventually develop, with the result that there is a typical bronchial cough, worse in the winter. The severity of the cough is not a measure of the activity of the tuberculosis. In advanced cases, with large cavities and gross infection, cough may be incessant and the sputum may be very profuse.

Tuberculous laryngitis is associated with cough which is husky and unproductive, and may be painful. Recurrent chills, colds, and attacks of bronchitis may lead

to cough which appears innocent in itself and yet is due to tuberculosis, and every patient with persistent bronchial catarrh should have the lungs properly investigated.

Eventually cough becomes a habit in many cases. The habit factor can be recognized by the excessive effort involved, which is quite disproportionate to the amount of sputum expelled. This type of cough can be voluntarily controlled by the patient.

The sputum in early cases is clear and mucoid. When there is cavitation it is purulent, and this is the type most commonly seen. The quantity varies from one or two drachms to many ounces. It must be remembered that in children, and in some adults, the sputum may be swallowed either accidentally or deliberately. Cough without apparent sputum does not exclude the possibility of lung disease, and in such cases the stomach contents or faeces should be examined for tubercle bacilli.

*Haemoptysis* may occur in the early stages of the disease as an occasional streaking which is neglected by the patient. On the other hand, there may be a more profuse haemorrhage when a vessel has been eroded. An early profuse haemoptysis may be fortunate for the patient as it may draw attention to an early lesion. In the later stages of the disease there may be even more profuse, and possibly fatal, haemoptysis from the rupture of an aneurysm in a cavity. This is the only dangerous type of haemoptysis in pulmonary tuberculosis for it may prove to be uncontrollable.

*Dyspnoea* is a frequent complaint, and it may be due to a variety of causes. When it comes on suddenly it may result from a spontaneous pneumothorax, or from rapid accumulation of fluid in the pleura. When it comes on gradually it is more likely to be due to progressive fibrosis and destruction of the lung tissue. In addition the toxic effect on the heart muscle, the anaemia, and the general asthenia all play a contributory part in producing dyspnoea. Sometimes dyspnoea is spasmodic; it may occur at night accompanied by wheeze, and the clinical condition simulates spasmodic asthma. Every patient with "asthma" should be investigated to exclude this possibility.

*Pain* is a common symptom which may be due either to muscle spasm, as a result of constant coughing, or to pleurisy. When it is remembered how common it is to find pleural



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tuberculosis is often followed by effusion which may become purulent.

*Pleural effusion* may occur as an initial event or as a complication of established pulmonary disease. Tuberculous empyema is comparatively rare. The pleural complications are more fully considered in the section on diseases of the pleura.

Tuberculous laryngitis is not uncommon. There are not always symptoms, and therefore a routine laryngoscopy should form part of the physical examination of the patient.

Tuberculous lesions are comparatively uncommon in the remainder of the respiratory tract. Occasionally an ulcer may form on the lip or tongue as a result of direct contact with infected sputum. This condition only occurs in patients who are rapidly losing ground. The tonsil is not very commonly affected. A persistent discharge from the ear, with indolent granulations on the drum, is sometimes seen; bacilli may be found in the discharge. The prognosis is not necessarily bad.

*Alimentary.*—It is inevitable that, with open pulmonary tuberculosis, bacilli will be swallowed in considerable numbers, and it is not surprising that intestinal tuberculosis is a frequent complication. The lesions are most commonly situated in the terminal part of the ileum, the caecum, and the ascending colon. The lymphoid follicles and Peyer's patches are congested and swollen at first, and later necrosis occurs, with ulceration. The ulcers are situated in the transverse axis of the bowel, they are deep and undermined, and may extend down to the peritoneum. On the peritoneal surface of the bowel tubercles and dilated lymphatics are seen.

The symptoms of intestinal tuberculosis are very diverse. There may be little or no abdominal discomfort with advanced disease, and, conversely, there may be symptoms which point plainly to the intestinal tract in cases in which at post-mortem there are no demonstrable lesions in any part of the bowel. Most commonly, in the early stages, there are slight digestive disturbances. Discomfort after meals, a feeling of fullness, occasional diarrhoea and constipation, are very common and may be so slight that their significance is overlooked. Commonly the patient feels unwell, as may be shown by an unexplained nervousness as to his condition, the weight falls, and there may be some pyrexia. A fairly characteristic feature is a tendency to remissions.

## PULMONARY TUBERCULOSIS

220

Pain is not by any means always present, nor is it an early symptom. It usually commences in the centre of the abdomen, but later radiates towards the right iliac fossa. It may be situated in the epigastrium and it may have such a definite relation to meals that the presence of a gastric ulcer is suspected. It is usually most severe in the afternoon. At a later stage the pain may be cramp-like or colicky. It is stated that a throbbing or burning sensation in the abdomen is associated with the presence of enlarged glands, and that this may occur even when the mass has completely calcified.

The reason for the pain is not clear. Severe ulceration may exist without any pain, and it is suggested that hypermotility and spasm are the usual causes. The pain is associated more with muscle irritation than with the actual ulceration, and this in turn is thought to result from inflammatory involvement of the nerves in Meissner's plexus, which is usually more involved than that of Auerbach.

*Diarrhoea*, although it may be regarded as the classical symptom, is by no means the rule, and there may even be constipation. The onset of diarrhoea is often gradual and it commonly follows constipation. A frequent feature is the occurrence of remissions during which the bowel action may be practically normal. In many cases there is alternating diarrhoea and constipation. The cause of this diarrhoea may be either the increased peristaltic movements of the bowel or diminished absorption of water as a result of interference with blood and lymph drainage. In some cases there may be very severe watery diarrhoea leading to dehydration, the "intestinal sweats" of Graves. When ulceration occurs in the small intestine the prominent feature may be either diarrhoea or constipation, but, with colitis, diarrhoea is the rule. It is stated that constipation is associated with ulceration at the hepatic flexure of the colon.

The number and situation of the ulcers bear no relation to the severity of the diarrhoea. This symptom may, in fact, be very marked when the only visible post-mortem change appears to be a simple catarrh of the mucous membrane. *Tenesmus* is only present when the rectum is involved. Other abdominal symptoms are *nausea* and *vomiting*. These are not very common, but they may add to the difficulties of diagnosis. They are usually associated with epigastric pain.

There may be considerable impairment of the appetite and distressing flatulence at times.

The *temperature* is very variable. In the majority of cases which prove fatal there is a high temperature which is usually remittent, but the patient may sometimes be afebrile throughout the whole course of the disease. It is necessary to consider the possibility of an abdominal complication when the temperature remains raised while the pulmonary signs appear to indicate quiescence.

In rare cases there may be a typhoidal picture, and the diagnosis may depend upon the serological reactions.

*Haemorrhage* of any gross degree is rare. It may occur either from the small or from the large bowel, and fatal cases have been known. As a rule, however, the intermittent passage of small quantities of blood is all that occurs.

The *weight* falls rapidly in most cases and an unexplained drop in weight has much the same diagnostic significance as unexplained pyrexia.

It must be remembered that many of the foregoing symptoms may be present without any demonstrable disease in the bowel. The symptoms are then thought to be toxic in origin and they undoubtedly result from a functional disturbance of the bowel mechanism. The existence of this type of symptom makes exact clinical diagnosis extremely difficult.

*Physical examination* is, as a rule, negative, and, beyond a localized tenderness in those cases in which the peritoneum is involved, there may be nothing to be felt. Glandular enlargement and the hypertrophic type of disease may cause palpable masses. Rigidity is very suggestive of perforation and local peritonitis. In most cases the diagnosis must depend upon a consideration of the symptoms, a suspicion of the presence of tubercle, and the results of special investigations.

The investigations are not very helpful. *Tubercle bacilli* can be found in the faeces in the absence of ulceration. X-ray examinations are of more value, for it is often possible to demonstrate localized areas of spasm in the region of the ulcers. When the colon is involved there may be a spastic filling defect which is usually permanent and suggests an organic alteration in the wall of the bowel. Adhesions, as well as chronic tuberculous ulcers, also may produce a permanent alteration in the outline of the normal barium shadow. "Intestinal hurry" is associated with active ulceration in the small bowel in some cases, but in atonic types of the disease and in early obstruction

## PULMONARY TUBERCULOSIS

231

there may be marked retardation of the progress of the meal. When the caecum is involved a barium enema may demonstrate constant spasm and the organ partially fills and empties; this condition of "intolerant caecum" appears to be fairly characteristic.

*Fistula-in-ano* is a complication of pulmonary tuberculosis which, while not very common, may present itself as an initial complaint. Any patient with a fistula should have the respiratory tract investigated with special care.

*Skeletal Tuberculosis.*—It is quite rare to find tuberculosis of bones or joints associated with pulmonary disease, but the association may occur. The spine and ribs are sometimes affected.

*Complications in the nervous system* are not common, although tuberculous meningitis is an occasional terminal event. Tuberculoma in the brain may occur as a complication of a primary infection in children.

A secondary anaemia may develop in chronic phthisis, but it is rarely an early symptom. Subacute or chronic arthritis is sometimes met with in cases of active pulmonary tuberculosis; the condition may closely simulate rheumatoid arthritis, and the joints have been known to flare up when a pneumothorax has been induced. It is probable that the joint lesions are not themselves tuberculous. Clubbing of the fingers and hypertrophic pulmonary osteoarthropathy are rare. Amyloid disease is occasionally present. The mentality of the phthisical patient is traditionally optimistic (spec phthisica) but depression is not rare. Symptoms suggestive of neurasthenia are common in the early stages and a toxic psychosis may cause the lung disease to be overlooked. The correct treatment of this type of case is the treatment of tuberculosis and not, as sometimes happens, incarceration in a mental hospital.

*Differential diagnosis.*—It must be remembered that a positive diagnosis should only be made after the demonstration of tubercle bacilli.

The chief pathological conditions which must be differentiated from pulmonary tuberculosis are:—

- (a) *Acute infections.*—Pneumonia of any sort may be mistaken for acute tuberculosis and vice versa. Persistent pyrexia is in favour of tubercle, although a concealed empyema may be responsible. Repeated sputum examinations will elucidate the diagnosis.
- (b) *Lung abscess.*—A chronic abscess is a cavity in the lung, and

the two conditions may be clinically indistinguishable. The sputum examination is the important investigation.

(u) *Chronic infection*.—Actinomycosis and streptothrix infections may present identical clinical appearances, but the sputum will be negative for tubercle and will almost certainly show the presence of the causal organism if properly examined.

(d) *Fibrosis of the lung*.—Fibrosis of the lung should always be regarded with suspicion. A history of some possible cause of the fibrosis may assist in the diagnosis, but it is not uncommon for tubercle to complicate some more innocent condition.

(e) *Bronchiectasis* may give rise to considerable difficulty, for it may precede or follow tuberculosis. Lipiodol will demonstrate the dilatation, but the sputum examination is necessary in order to confirm or exclude tubercle. This is particularly important because in some cases, especially of congenital cystic bronchiectasis, the lesion may be situated in an upper lobe and the physical signs may be suggestive of tuberculosis; such patients not uncommonly find their way to a sanatorium in time, which is bad for them psychologically, and may be risky. A patient who has physical signs suggestive of tubercle but a persistently negative sputum should have a bronchogram done (fig. 17).

(f) *Pulmonary syphilis*.—This is so rare that it is hardly a source of error. The Wassermann reaction should always be done in any patient who has an obscure chest complaint and a persistently negative sputum.

(g) *Bronchial carcinoma*.—Tubercle and growth may coexist, so that a positive sputum does not exclude the possibility of a bronchial carcinoma, and a bronchoscopy should always be done if the symptoms and signs are at all suggestive of malignant disease, especially if the patient does not respond to treatment.

(h) *Haemoptysis* of unexplained origin is commonly due to tuberculosis, but it also occurs in heart disease, especially in mitral stenosis. A careful examination of the heart is therefore indicated when haemoptysis is a prominent symptom.

(i) The patient who presents a picture of primary pleural effusion may have tuberculosis or a growth. Careful examination of the fluid, including culture and guinea-pig inoculations, may establish a diagnosis of tuberculosis. The fluid may be removed and a further X-ray may demonstrate the lung lesion, or an air replacement may be done, and a thoracoscopy may then show tubercles or malignant nodules in the pleura.

*Prognosis*.—It is necessary to be clear about the terms applied to the different stages of tuberculosis. So often the term "cure" is used unjustifiably and the patient is lulled into a false sense of security.

Active tuberculosis is usually obvious, but sometimes there

is a doubt. It may be defined as "a tuberculous lesion which is considered to be responsible for toxæmia, or which can be shown on serial skiagrams to be altering". It does not matter in which direction X-ray changes occur, for continued improvement is proof that the lesion is not yet fully quiescent.

*Quiescent tuberculosis* is the term applied when there is no evidence of toxæmia and the X-ray changes are stationary. Relapse is still possible if the patient is careless or unfortunate.

*Arrested tuberculosis* should indicate that the lesion is soundly healed. There is no sharp distinction between quiescence and arrest and, in practice, the disease is presumed to be arrested when it is known to have been quiescent for two years or more. It has been well said that it is unwise ever to give a dogmatic prognosis in pulmonary tuberculosis. Unfortunately the patient and relatives are often, quite rightly from their point of view, insistent that an attempt should be made to forecast the probable duration and result of the illness, and therefore the evidence on which the prognosis is based must be considered.

The whole outcome depends upon the balance between the resistance of the patient and the virulence of the organism; this latter factor is difficult or impossible to assess and therefore the former factor must receive chief consideration upon the following lines:—

(a) *The general appearance of the patient.*—The patient who "looks the part", clear-skinned, rufus, or fair and blue-eyed, of delicate build, and with a phthisical type of chest, is one who is not likely to put up a good fight.

(b) *The age and sex.*—These are of some importance, for it is found that adolescents and young adults, especially females, have a relatively low resistance, and there is a definitely increased mortality between the ages of 15 and 25 years.

(c) In particular, certain races, Highlanders, Irish, and those from sparsely populated rural districts, are known to have a deficient resistance to the infection.

(d) *The previous history* of the patient, and his known ability to resist other infections, must be taken into consideration although it is not of primary importance, and the *family history* is sometimes of value if it is clearly established that other members of a family have proved unable to overcome the disease.

(e) Far more important is the *environment* of the patient. Those who can afford to spend time and, to a less extent, money will obviously stand a better chance of becoming quiescent, whereas



those who, for family or financial reasons, have to return to work before they are really fit are likely soon to break down.

In assessing the influence of environment upon prognosis it is desirable to remember not only the home conditions and the conditions in which the patient will have to work, but also the amount of travelling which will have to be done, for this is the factor which frequently tends to produce physical and nervous exhaustion and thus lowers vitality.

(f) The extent of the disease must be taken into account. Certain classifications have been made with a view to assisting in the determination of the prognosis. Most of these are based upon the extent of the disease at the time of observation. Perhaps the best known is the Turban-Gerhardt classification, which is as follows :—

*Stage 1.* Apical lesion only ; physical signs not below the 2nd rib.

*Stage 2.* Physical signs, if unilateral, not below the 4th rib ; if bilateral, not below 2nd ribs ; no cavity formation.

*Stage 3.* Includes all cases with more extensive disease.

The Ministry of Health has adopted the following classification for use in the annual returns :—

Class A. Tubercle bacilli not found.

Class B. Tubercle bacilli present.

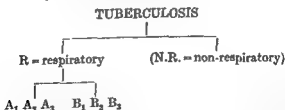
These are each subdivided into three groups :—

Group 1. Slight constitutional disturbance and few signs or radiological changes.

Group 2. Cases which are not included in groups 1 or 3.

Group 3. Profound systemic disturbance or constitutional deterioration, and pronounced impairment of function.

The Scheme may be tabulated as follows :—



An uncomplicated pleural effusion for which no other cause can be found should be regarded as tuberculous and the case placed in group 1 of class A or B according to the bacteriological report on the fluid.

Exact classifications are not, in fact, very helpful, because it is natural to expect that, the more extensive the disease, the worse the prognosis. Yet the resistance of the patient is so important

that some will overcome an extensive infection comparatively well, whereas others who have a mild lesion at first, but poor resistance, may lose ground rapidly without any response to treatment. It is better, therefore, not to depend upon the extent of the infection, but rather to take a view of the progress of the patient which is based upon observation over a period of six months or more.

(g) As a corollary it follows that the response of the patient to treatment while under observation can prove a most valuable guide to the prognosis. In determining the prognosis the temperature curve, the pulse rate, the weight, the persistence of bacilli in the sputum, and the alteration in the radiographic appearances must be considered together.

(h) The presence of complications will obviously prejudice the prognosis.

(i) The sedimentation rate is of value in prognosis, for a steady fall in the rate, when repeated observations are made, is evidence of healing. The results, however, must be taken in conjunction with the clinical state.

(j) The relation of pulmonary tuberculosis to pregnancy may be appropriately considered here. A woman may feel better during pregnancy, but a complete breakdown is likely during lactation. A pregnancy in a woman with active phthisis should always be terminated before the twelfth week. A more difficult decision has often to be made when considering the question of permitting a pregnancy to proceed in a woman who appears to have reached a quiescent stage. Each case should be judged on its merits, but, as a rule, it is unwise to allow a pregnancy to proceed to term until the sputum has been negative for four or five years. There can never be cause to regret following this procedure, whereas disregard of this rule has led to many tragedies.

It will be seen, therefore, that many points have to be taken into consideration in framing a prognosis. It is always a matter of great difficulty, and the physician should never be surprised if his forecast should turn out to be inaccurate in either direction.

## CHAPTER 28

### PULMONARY TUBERCULOSIS (*continued*)

#### Treatment

THERE is no curative treatment for phthisis. The best that we can do is to place the patient in the most suitable surroundings for his own immunity processes to develop. Rest, good food, fresh air are all of material assistance in attaining this end, and the education of the patient to live a restricted life within the limits of his capacity is of supreme importance.

Treatment may be considered in three stages.

1. *The initial stage.*—The chief indication at this stage is rest, both physical and mental. The duration varies in different cases. The following stages of rest, laid down by Wingfield, form a sound basis.

(i) No physical nor mental exertion of any kind. Very careful nursing.

(ii) The patient is allowed to talk and to feed himself.

(iii) Recumbency, with short periods of semi-recumbency. The patient is allowed to wash and feed himself.

(iv) The amount of time during which the patient is allowed to sit up in bed is gradually increased.

(v) The patient is allowed out of bed, at first for half an hour and later for increasing periods, in a chair.

(vi) The patient is allowed to walk for a short distance in the room.

(vii) The patient is allowed to wash out of bed, to bath, and the amount of walking is increased.

Freshly diagnosed cases should have a minimum period of two months in bed. At this stage of treatment relapses are very common if the patient is allowed to make unduly rapid progress. The patient with early disease and slight fever should usually start at stage iii, and his progress should be carefully watched for a week or ten days. Should the condition be satisfactory the scale may be gradually ascended.

The early case without fever may start at stage iv or v, being carefully watched.

## PULMONARY TUBERCULOSIS

237

*The main indication for rest is toxæmia.* The general appearance of the patient, the temperature, the pulse rate, and the weight are the most valuable guides in this respect.

Such strict treatment is often irksome to the patient, especially if, as often happens, he does not feel very ill and his symptoms subside rapidly. It is absolutely necessary to have the close co-operation of the patient throughout the period of treatment. Those who break rules seldom do any good, and it is advisable that this should be explained to the patient before treatment of any sort is undertaken.

From the practical point of view it is much better for this primary period of rest to be undertaken in a hospital or some such institution. It is only in exceptional circumstances, with quite unusual home facilities, that the patient should be allowed treatment at home. The discipline of an institution is a good introduction to the type of life which the patient will have to lead for a considerable period, perhaps for the rest of his life. The febrile case is not as a rule suitable for admission to a sanatorium.

2. *The sanatorium stage.*—The development of the sanatorium system has undoubtedly been to a considerable extent responsible for the improvement in the results of treatment. It is most important, however, that sanatorium treatment should be reserved for those cases for which it is indicated, and that unsuitable cases, or cases at the wrong stage of the disease, should not be sent.

The proper cases for sanatorium treatment are patients who have had a period of at least two months' rest in bed. There should be practically no evidence of toxæmia when the patient is allowed up. The pulmonary lesions should not be sufficiently extensive to incapacitate the patient, or to render it unlikely that he will be reasonably efficient on discharge, and there should be no major complications present. While the ideal case should fulfil these requirements, it is a well-known fact that a considerable proportion of patients admitted to sanatoria do not in any way conform to these criteria. If sanatorium treatment were strictly confined to suitable cases there can be no doubt that the results would be still better. It is necessary for both the doctor and the patient to understand exactly what it is that the sanatorium is in a position to do. In the first place, and perhaps most important of all, the patient is subjected to a physical and mental routine which has the ob-

co-ordinating his rest and exercise, and bringing them to the most suitable pitch, within the limits of his capacity, so that he may return to his occupation and lead a useful, though restricted, life. In other words, *a most important function of the sanatorium is education of the patient.* In addition, his general resistance is built up by suitable hygienic measures, sun, fresh air, good food. The patient is taught to take care of himself and to avoid the risk of infecting others. In addition to this the various lines of special treatment are carried out in the sanatorium.

The principle of graduated exercise is carried on in exactly the same way as in the initial stage. The patient is at first allowed to walk increasing distances, starting with quarter of a mile and increasing to three miles or more, at first on the flat, and later up hill. The temperature is taken after half an hour of rest; any increase indicates that the patient is doing too much. Still later outdoor work is undertaken, usually in a garden, starting at first with light tasks and gradually increasing until the patient is able to undertake moderate physical work without reaction.

The criterion by which progress is judged, and which determines whether the patient is fit to increase his activities, is the absence of toxæmia. The temperature, the pulse, and the weight are of greatest importance, and the symptoms of local disease, cough, and sputum are less significant. Increase in physical exercise sometimes leads to transient hæmoptysis, which is not necessarily a symptom of increased activity of the disease. Physical examination of the chest is perhaps less helpful than any other method of assessment of progress, for spread of the disease shows itself sooner by toxic symptoms than by change in the physical signs.

The theory on which gradual increase of exercise is based is that exertion leads to some degree of auto-inoculation with tuberculin, and that this process, so long as it is not allowed to get out of hand, reacts beneficially upon the patient. The theory is probably quite sound. It follows that very careful temperature records must always be kept of patients while they are under treatment.

The type of sanatorium to be selected depends upon many factors, of which the chief are the financial position of the patient and his individual preference. So long as the sanatorium is so situated that the maximum of sun and fresh air

can be obtained, the question of climate is of relatively little importance. There is a general consensus of opinion that there is little to be gained from sending patients abroad, and that the sanatoria in this country are as suitable as any. The patient who feels that there is special virtue in a foreign climate may well be permitted to go there, as his faith is likely materially to assist his recovery. It is to be remembered that excessive heat and direct exposure to sun are more to be avoided than a cold climate, and that a dusty atmosphere is pernicious.

The patient whose finances permit may choose one of the more luxurious sanatoria. From the point of view of his health this is by no means a good thing, as such patients are less likely to concentrate on the primary object of their visit to the sanatorium, which is to get well.

It must also be remembered that residence in a sanatorium is open to the objection that it may breed a neurosis which is very difficult to overcome. The habit of patients of discussing their complications and treatment with others is a disadvantage, and efforts must be made so far as possible to overcome this and to encourage an optimistic outlook. The patient who is cheerful and confident starts with a great advantage and has a much better chance of doing well.

3. *The quiescent stage.*—By the time that the patient has left the sanatorium and is fit to return home, his disease should have become quiescent. It does not necessarily follow that tubercle bacilli will have disappeared from the sputum, or that there will be no physical signs of disease in the lung, but it does mean that the resistance of the individual is sufficient to keep the infection in check. The patient must carry out what has been taught in the sanatorium, and must look after himself and guard himself from minor infections. He must keep under medical supervision for years, often for the rest of his life, and skiagrams should be taken at intervals. It is a fact that many patients, after they have left the sanatorium, tend to forget what they have learnt and to overtax their strength, with the almost inevitable result of a bad relapse. In this respect again, it is an astonishing feature that those patients who are financially better off take less care of themselves, with the result that their chances of becoming stabilized are actually less than those of patients who are less fortunately placed.

Although it is customary to regard sunshine as good for

tubercle, it is necessary to be very cautious in the application of this therapy to cases of phthisis. Direct sunlight and ultra-violet light are very apt to provoke *haemoptysis*. This applies not only to cases of active tubercle but also to latent lesions. Sun-bathing in general is therefore unwise, unless it is certain that the lungs are healthy, and the indiscriminate use of ultra-violet rays as a tonic should be condemned.

There is no doubt that sunshine and ultra-violet light are both beneficial in certain circumstances, but every care should be taken to exclude the presence of even minute tuberculous foci in the lung before any individual is encouraged to indulge in this type of ray therapy.

The further treatment of pulmonary tuberculosis may be divided into the following groups :—

1. Attempts to build up resistance of the patient by medicinal means.
2. Attempts to destroy the tubercle bacillus.
3. Special measures to secure rest of the affected lung.
4. Treatment designed to relieve symptoms and to prevent complications.

Medicinal treatment has proved most disappointing. It is customary to give cod liver oil, with or without malt extract, and the universal use of this substance can be taken to mean that it has a beneficial action, although the effect is by no means pronounced. Fat undoubtedly improves nutrition and helps the patient to put on weight, and the vitamin content probably has some value in increasing resistance to infection. Another drug which has been extensively employed in an attempt to promote healing is calcium. At first sight it would appear that this should be good treatment, for the soundest healing which can be achieved is by calcification of the lesions. There is, however, ample calcium in the body already, and it is doubtful whether there is any need for a further supply. Calcium lactate given by the mouth is practically useless, and it is only too probable that calcium salts given intramuscularly or intravenously have very little effect. From time immemorial it has been customary to give garlic and other oils; creosote has also been given. There is no evidence that any of these have any value.

Since the discovery of the tubercle bacillus repeated attempts have been made to immunize patients by giving injections of

**Tuberculin.** The value of this treatment has been hotly disputed, but the great majority of observers are not convinced that it has any beneficial effect.

The only method which is at present employed in an attempt to destroy the bacillus is the administration of gold salts by injection. After nearly twenty years' experience there is still no general agreement about its value, and its mode of action is still uncertain.

It is probable that gold destroys some of the bacilli but the reactions which follow the injections are not due simply to the absorption of the products of disintegration of the tubercle bacillus; many of the features of the reaction are the result of poisoning by the metal and therefore can in no way be beneficial.

Gold is most commonly given in order to assist in the absorption of recent exudative lesions in the lung; it is not generally considered to be helpful when there is much fibrosis. Sometimes it is used when there is a little active disease in the lung opposite to that which is being treated by pneumothorax. It must be stated, however, that many physicians do not consider that gold has any demonstrable therapeutic action at all.

The prototype is Sanocrysin or Crisalbine, a white crystalline solid which is a double thiosulphate of gold and sodium. The injections are given intravenously and it is customary to begin with 0.05 gram, giving injections at intervals of about 5 days, and to increase the dose fairly rapidly until 0.5 gram is being given. A full course consists of about 4 grams administered in the space of two months. Higher doses than 0.5 gram are sometimes given if the physical condition of the patient is good, and if there are no reactions. Myocrysin (thiomalate of gold and sodium) is a similar preparation which is suitable for intramuscular injection.

Gold should not be given to any patient who has any suggestion of kidney damage.

The reactions which may follow gold injections are very characteristic. There is exacerbation of fever, with albuminuria and red cells in the urine. There may be a skin eruption which varies from an erythema to an acute exfoliative dermatitis. Gastro-intestinal symptoms, nausea, vomiting, and diarrhoea, which are characteristic of poisoning with heavy metals, are



sometimes evident. There may be some ulceration of the throat which is not serious as a rule although, in severe cases, it may form part of the picture of an agranulocytosis, a condition which is usually fatal.

*Streptomycin*.—Encouraging results are reported from the use of this drug, but it is not generally available in this country at present. In any case, a considerable time must elapse before the effect of treatment can be assessed. It would appear, however, that the margin of safety is small and that great care must be exercised in its administration.

### *Collapse Therapy*

The great advance of this century in the treatment of chest disease has been the introduction of collapse therapy. It is possible to rest the affected lung, or a part of the lung, and the collapse may be made temporary or permanent at will. The great value of collapse treatment is that it enables cavities to be closed with speed and certainty. Collapse may be achieved by three main methods, by *pneumothorax*, by *phrenic paralysis*, or by *thoracoplasty*.

*Artificial pneumothorax*.—It is now accepted that temporary collapse of the lung by this means is of very great benefit in promoting healing. The indications are as follow :—

(a) In cases where there is early infiltration in one lung which is not responding to routine treatment by rest.

(b) In cases where there is a cavity which fails to diminish in size. Recent active cavities may heal of their own accord and therefore such a condition is not necessarily an indication for pneumothorax although, when there is any doubt, it is better to collapse the lung early. It must be remembered that, if pneumothorax is delayed too long, there is a possibility that adhesions may form.

(c) The presence of some complication, such as laryngitis or enteritis, is an indication for the induction of a pneumothorax when the disease is more or less confined to one lung.

(d) A pneumothorax may be induced in order to control a profuse haemoptysis. In such cases it is necessary to be absolutely certain from which side the blood is coming.

(e) Bilateral pneumothorax is sometimes used in the treatment of cavities in both lungs.

*Pneumothorax Technique*

*Pneumothorax apparatus.*—Air is introduced into the pleural cavity by means of a special needle which is inserted through an intercostal space. There are two main types of apparatus in use. In the first the air is supplied from a cylinder by the pressure of a piston. In the second water is allowed to syphon from one bottle into another so that air is expelled through rubber tubing to the needle in the chest. Two types of apparatus are described.

1. The apparatus (fig. 1) devised by the author consists of four essential parts: the pump, the regulating valve, the manometer, and the filter. The use of an aneroid manometer enables the apparatus to be contained in an exceptionally small case. The pump has a capacity of 200 c.cs., and is made entirely of metal. The combined weight of the piston, piston-rod, and two-way cock is calculated to produce a gas pressure equal to that of a column of water 35 cms. in height. The piston-rod is hollow, and is fitted with a two-way cock of convenient shape to act as a handle by which the piston is pulled up. One end of this cock terminates in a screw cap for the purpose of holding a cotton-wool filter for the atmospheric air. The opposite end is bent downward, and is joined to the regulating valve by rubber tubing. By means of this valve the rate of flow of air to the pleura can be controlled with great ease and accuracy, or it can be cut off entirely. From the outlet port of the regulating valve the tube divides into two branches. One leads via the filter to the patient, and the other to the manometer. The quantity of air which has passed to the chest is indicated on the outside of the piston-rod by a graduated scale. The filter consists of a glass tube with a metal cone at one end and a screw cap at the other. The cone fits into a counterpart, which is part of the "T" conjunction connecting the manometer and filter to the regulating valve.

The advantages of this instrument are that it is readily portable, there is little likelihood of accidental damage, it is always ready for use, and the difficulties inseparable from the use of liquid in the manometer are obviated. When used for a refill the instrument is placed upright, the cylinder is filled with atmospheric air, and the flow to the chest is regulated by the valve, so that any desired pressure can be maintained; the

scapula, and the mid-axilla. The skin is first cleaned and 2 per cent novocaine is injected into the skin and into the parietal pleura, using rather more than for a simple puncture of the chest. A preliminary injection of morphia is only necessary in very nervous patients.

The trocar and cannula are then connected to the apparatus in the ordinary way, and the tube is pushed firmly and slowly onwards through the intercostal space; there is rarely any need to incise the skin. With the ordinary type of trocar and cannula, which is not provided with a groove, there is no indication when the pleural space is reached and the estimation of the position of the needle becomes a matter of guess-work. The trocar must be withdrawn almost to the end of the cannula at intervals, and a negative swing in the manometer shows when the cannula is in the pleural space. Should the cannula have penetrated the lung, there is usually a negative and positive swing which indicates atmospheric pressure and, should there be many adhesions, there may be no movement on the manometer at all. Sometimes the cannula injures a blood vessel, in which case blood will be seen in the small glass window in the rubber tubing. When the grooved trocar is used the manometer shows a negative kick as soon as the pleural space is reached, and the position of the instrument can therefore be much more readily ascertained. When it is certain that the end of the cannula is in the pleural space the trocar is withdrawn and the extent of the negative swing is noted. Air in the cylinder is free to flow into the chest. The lung, being an elastic structure, tends to contract and therefore to draw air into the pleural cavity, and in many cases it is amazing to note how much air can thus be drawn in by simple suction. When bottles are being used, it is found that the pressure bottle must be raised at intervals, as the pressure in the gas bottle tends to become too low in view of the column of water which has to be supported, but when the cylinder is being used no adjustment is necessary except to refill the cylinder when the whole quantity of air has been withdrawn into the chest. The amount of air which is allowed to enter at an induction varies according to the size of the pleural cavity, and the presence and extent of adhesions, but, on the average, it is found that 400 c.cs. is a satisfactory quantity. The pressures should be left slightly negative.

The cannula is removed at the end of the operation and a collodion seal is applied to the puncture. The patient should be instructed to cough as little as possible for the next few hours as otherwise there is a risk of surgical emphysema developing. This is not a very serious event but it means that there is some loss of air from the pleural cavity.

When the induction has been satisfactory, it is customary to give a refill on the first or second day and then to aim at a total of three refills in the first week, two in the second and one in the third. The spacing of the refills depends entirely upon the individual patient.

It is not usually desirable to screen the chest or to take a film until at least 1600 c.cs. of air have been admitted into the chest, as very little change is likely to be seen before this.

*Pneumothorax refill.*—The technique of a refill is very simple. Novocaine should always be injected, both into the skin and down to the parietal pleura; this minimizes the risk of collapse from pleural shock. It is necessary to see that the manometer registers a free swing after the needle is inserted, before any air is introduced, otherwise air may be injected into the subcutaneous tissue, into the lung, or even into a blood vessel. Complications, such as air embolism, are very rare if this precaution is observed although they may still occur.

The air is allowed to enter at a moderate pressure, usually about +20 cms. of water, and the quantity admitted and the final pressures vary in different patients. The process must invariably be controlled by screening the chest at frequent intervals, preferably after each refill. It is only by doing this that the state of the collapse, changes in the length of the adhesions, the position of the mediastinum, and the presence of fluid can be properly observed.

In some cases, without any apparent reason, there may be febrile reactions after the refills and eventually fluid may form in the pleura. This effusion is an ordinary exudate and it is not necessarily detrimental to the patient. In most cases it is wiser not to remove the fluid unless there is so much that the mediastinum is displaced. When there is only a moderate effusion it may be left alone, and it is found that in such cases the sound lobe expands and the fluid becomes encysted opposite the damaged area, thus ensuring collapse of that part of the

lung for a period of years. This encysted effusion resembles an *oleothorax* (fig. 48) in its action, and further refills of air may become unnecessary. Infection of this fluid, with the formation of empyema, is rare.

Sometimes air may gain entrance to a radicle of the pulmonary vein, causing an air embolus which may affect the brain, and which may be fatal. This accident should rarely occur if precautions are taken to see that the manometer is swinging freely before any air is allowed to flow into the chest.

An occasional complication is *spontaneous pneumothorax*. This usually results from the rupture of adhesions when the intrapleural pressure is high. There are sudden pain in the chest and *dyspnoea*, and the mediastinum is often found to have shifted. The accident should be suspected from the symptoms and signs, and confirmation can be obtained on the screen. It is not often necessary to adopt special treatment but, when there is much discomfort, a sufficient quantity of air can be withdrawn from the chest. Empyema may follow.

In many cases it is found that adhesions are present even although the pneumothorax has been induced quite easily (fig. 50). This is only natural when it is remembered that there is almost invariably an area of pleurisy overlying the lesion, but it means that the damaged part of the lung is therefore the very part which cannot collapse. In order to overcome the difficulties which arise from the presence of adhesions, which would otherwise render a great many pneumothoraces completely useless, a great deal of attention has been paid to the technique of division of adhesions. This is done by endoscopic methods and the results are now very satisfactory. A thoracoscope is inserted through the intercostal space and the adhesion can be seen quite clearly. In the method of Jacobaeus two punctures are necessary, for one instrument carries the light and another carries a cautery. In the thoracoscope devised by Chandler the complete operation can be carried out through the one tube, and the discomfort of the patient is therefore minimized. The good results of pneumothorax treatment depend on successful adhesion cutting in many cases.

*Duration of pneumothorax treatment.*—The interval between refills varies in different patients. Some physicians prefer to give small quantities at fairly frequent intervals, e.g. one week, whereas others give larger quantities at intervals of two, three,

or four weeks. The best interval is decided by screening the patient before the refill.

The lung should be collapsed sufficiently long to allow the lesion to heal. Duration of treatment should not as a rule be less than three years, and it may be as long as five years. When it is decided to abandon treatment films should be taken at intervals of a few weeks in order to check the state of the expanding lung. If there be the slightest suggestion of active disease the treatment can be resumed. After the lung has finally expanded it becomes firmly adherent to the chest wall in most cases and, should there be any return of activity, it will quite likely be impossible to re-induce the pneumothorax.

*Tension cavity.*—Sometimes a successful pneumothorax fails to close a cavity and, in fact, the cavity may even appear to enlarge after the pneumothorax is established. The cause of this "tension cavity" is a partial obstruction of the bronchus draining the cavity so that a check-valve (p. 163) effect is produced. When it is certain that a tension cavity is present the pneumothorax should be abandoned. The best method of treating a tension cavity is not yet agreed, and several different opinions are held, but it is not within the scope of this book to consider them.

*Oleothonax.*—This term is used to indicate the replacement of air by oil in an artificial pneumothorax in cases where, for any reason, it is inexpedient to continue giving refills. The advantage is that the patient is independent of refills, but there are certain disadvantages, notably that the collapse produced is more or less permanent, and that some such accident as rupture of the oleothorax may occur, and this is an event which is sufficiently common to make it necessary to weigh the risk carefully before deciding upon this form of treatment.

The chief indication for oleothorax is commencing obliterative pleurisy. A dense sheet of adhesions develops at the base and rapidly spreads upwards, causing the lung to expand. The replacement of air by oil interposes an effective barrier to the spread of the adhesions and enables the collapse to be continued. Oleothorax is also useful in patients who have to travel a great deal, or who are likely to be going for a long voyage, or to be living in regions where refills are not readily obtainable. It may sometimes be done in patients who are nervous of refills, or when there are repeated and severe pleural reactions. If kept for suitable cases this is a useful method of treatment.

Before oleothorax is even considered, the pneumothorax must be well established. The simplest substance to use is sterile olive oil which must be injected with strict aseptic precautions. It is customary to introduce from 200 to 400 c.cs. at a time, giving 800 to 1000 c.cs. altogether. The intrapleural pressure is registered at the same time and air is gradually withdrawn as the oil flows in. The process may need to be repeated once or twice. The cavity should never be entirely filled with oil as the resulting pressure may cause rupture of the lung; it is better to leave it about half full. The residual air is gradually absorbed and some expansion of the underlying lung takes place, so that eventually a circumscribed cavity, completely filled with oil, is left (fig. 48). The curious feature of oleothorax is that, when basal pleurisy is present, the oil is gradually pushed up until it is the upper lobe which is collapsed, and this is fortunate, for it is usually this region which is the site of the disease.

When this method is used there are comparatively few complications, although occasionally a pleural reaction may cause the temperature to be elevated for several weeks. Infection as a result of the introduction of organisms from outside should never occur but sometimes, if too much oil has been introduced, the pressure may be so great that the lung may be ruptured and infection may spread from the lung.

*Phrenic avulsion.*—The diaphragm may be paralysed, either permanently or temporarily, by operation on the phrenic nerve. A temporary paralysis is usually caused by crushing or cutting the nerve, whereas a permanent result is likely when two or more inches of nerve are avulsed. The indications for phrenic paralysis are as follow :—

1. Basal tuberculous cavities often respond well.

2. When there is an apical cavity, with gross surrounding fibrosis and evidence of tension in the longitudinal plane, presuming that a pneumothorax cannot be obtained. Apical cavities not infrequently heal, or become smaller, when the inspiratory tension caused by the descent of the diaphragm is abolished (figs. 59, 60).

3. In some cases of gross fibrosis with displacement of the mediastinum a phrenic paralysis is very useful in relieving the tension on the affected side of the chest.

4. Sometimes the phrenic nerve is paralysed as a preliminary to thoracoplasty. It must be remembered that when the diaphragm is permanently paralyzed any subsequent thoracoplasty must be complete, as a partial operation on the upper six or seven ribs merely leaves a functionless and potentially dangerous lower lobe when the diaphragm is out of action.

5. The diaphragm may be paralysed in cases where there is already a pneumothorax in order still further to collapse the lung.

It must be remembered that the intrapleural pressure will alter considerably after the diaphragm has risen and that large refills with a high intrapleural pressure, may actually invert the paralysed muscle. Great care must therefore be taken with the refills.

A temporary phrenic paralysis is usually effective for five or six months. It is commonly done in order to determine whether the lesion is likely to respond to this form of treatment for, if so, a permanent paralysis can then be carried out. Sometimes it is done merely as a temporary expedient to assist healing.

*Thoracoplasty*—This is the best procedure for obtaining permanent collapse of a hopelessly damaged lung. The operation can be carried out in one, two, or three stages, according to the general condition of the patient. It is usually reserved for cases with extensive chronic disease, in which a pneumothorax cannot be induced, or in which it has failed to produce improvement. Although it is a severe operation the mortality has diminished in a striking manner of recent years and thoracoplasty has been responsible for the recovery of a measure of health in a great many cases. It is, however, the last resort in collapse therapy and should only be undertaken after very careful consideration, and after prolonged observation of the response of the patient to other forms of treatment (fig. 44).

*Suction drainage of cavity.*—(Monaldi's method) Large chronic cavities are often not responsive to any method of collapse treatment and, in particular, thoracoplasty may fail to secure closure. The bronchial drainage of such a cavity is often very bad and continuous suction is found to be valuable in reducing its size considerably. It rarely happens that the cavity entirely disappears, but at least the condition is so improved that thoracoplasty is likely to succeed.

Steps are first taken to make sure that the overlying pleura is firmly adherent, or to cause adhesions to form (p. 268), and a thick rubber tube is inserted through an intercostal space into the cavity. The tube is then connected to a pump and gentle suction is applied. This may be kept up until X-rays show that the cavity has ceased to contract.

Other methods of collapse have been tried without proving very useful. Local collapse of a portion of the lung is sometimes achieved by open operation and filling of the extrapleural space with wax, fat, or a muscle graft. The chance of sepsis in these operations is considerable and the benefit to be gained is not worth the risk. Chronic disease in an upper lobe, with adherent pleura, is sometimes



treated by "extra-pleural pneumothorax". In order to produce this the parietal pleura is stripped from the chest wall and the lung is collapsed inwards so that an air space is formed between the chest wall and the two layers of adherent pleura; refills are then given in the ordinary way. It is too early as yet to assess the value of this procedure.

*Pneumoperitoneum.*—For many years it had been noticed that pregnant women with chronic phthisis showed clinical improvement during the last few months of their pregnancy. This improvement was not maintained, and delivery was often followed by a rapid spread of the disease within a few weeks. Eventually it was suspected that this ill-effect of pregnancy is due to the rapid descent of the diaphragm which follows immediately on delivery, and it was therefore assumed that the clinical improvement in the last few months of pregnancy is the result of the rise in the diaphragm, which has the effect of restricting the respiratory movements, resting both lungs to a limited extent.

The first application of this theory was the induction of a pneumoperitoneum immediately after delivery, in order to allow the diaphragm to descend slowly and so avoid sudden strain on the damaged lungs. It was found that the disease did not show an exacerbation in many cases when this was done. The logical consequence of the good results reported from this treatment was the induction of pneumoperitoneum in non-pregnant cases. The diaphragm can be raised three or four inches by repeated injections of air into the peritoneal cavity and the inspiratory excursion can be diminished accordingly. Pneumoperitoneum finds its main application when both lungs are involved. It can be combined with a phrenic crush in the treatment of an apical cavity when the opposite lung is diseased. Pneumoperitoneum is not a substitute for a pneumothorax. The actual management of a pneumoperitoneum is not altogether easy from a technical point of view, and the refills present considerable difficulties as compared with refills into the pleura. It is decidedly a method for the expert and it benefits comparatively few cases.

### *The Treatment of Prominent Symptoms*

*Cough.*—A moderate cough, which is productive, needs no medicinal treatment. When the cough is out of proportion to the

sputum produced, especially in the morning, a simple saline draught may prove to be effective :

|               |   |   |   |   |   |                        |
|---------------|---|---|---|---|---|------------------------|
| Sod. bic.     | . | . | . | . | . | 15 grains              |
| Sod. chlor.   | . | . | . | . | . | 5 grains               |
| Spt. chlorof. | . | . | . | . | . | 5 minims               |
| Aq. anisi     | . | . | . | . | . | ad $\frac{1}{2}$ ounce |

Lozenges such as troch. glycyrrhizae are valuable :

|                   |   |   |   |   |   |                     |
|-------------------|---|---|---|---|---|---------------------|
| Ext. glycyrrhizae | . | . | . | . | . | 3 grains            |
| Olei anisi        | . | . | . | . | . | $\frac{1}{2}$ minim |
| Troch. acaciae    | . | . | . | . | . | 10 grains           |

An irritating and useless cough may require a stronger sedative such as

|                    |   |   |   |   |   |              |
|--------------------|---|---|---|---|---|--------------|
| Syr. codein. phos. | . | . | . | . | . | 20 minims    |
| Syr. aurant.       | . | . | . | . | . | 20 minims    |
| Spt. chlorof.      | . | . | . | . | . | 2 minims     |
| Glyc.              | . | . | . | . | . | ad 60 minims |

or

|                 |   |   |   |   |   |           |
|-----------------|---|---|---|---|---|-----------|
| Tr. opii camph. | . | . | . | . | . | 20 minims |
| Oxymel scillae  | . | . | . | . | . | 20 minims |
| Syr. tolut.     | . | . | . | . | . | 20 minims |

or, if the cough proves intractable, the linctus heroin. et terpin chlor. (B.P.C.)

It must be remembered that in many cases the cough becomes a habit after a time. The patient must be encouraged to refrain from unnecessary coughing.

It is not necessary to stop most patients from smoking if they have little cough. The chief indications for forbidding tobacco are *associated catarrh*, which is a frequent factor in causing a chronic cough, and *laryngitis*. There is no doubt that intractable cough will often vanish within a few days of ceasing to smoke.

*Haemoptysis* — Minor degrees of blood-spitting are very common and their treatment is the routine treatment of pulmonary tuberculosis. Severe haemoptysis requires special treatment. Complete rest is the most effective measure, and the patient should be permitted to adopt the posture which gives greatest comfort. There is always a factor of mental distress and therefore reassurance of the patient and the avoidance of any appearance of concern are important, especially as haemoptysis is rarely fatal unless a large vessel has

breathing and rales. When the fibrosis is on the right side the displacement of the right upper bronchus may cause bronchial breathing to be heard over the upper lobe, and this may lead to an erroneous suspicion of a cavity.

The investigations in cases of massive fibrosis are relatively simple.

1. The sputum must be examined for tubercle bacilli.
2. An X-ray shows characteristic appearances. The lung fields are less translucent than normal, the heart and mediastinum are displaced towards the affected side, and the diaphragm may be raised (fig. 14). In addition there may be evidence of the causal condition, such as calcification or cavitation. Bronchial dilatation may not be evident in the plain film and a lipiodol examination is often desirable in order to determine the presence of bronchiectasis.
3. Should there be any suspicion of bronchial obstruction a bronchoscopy must be done.
4. In obscure cases the Wassermann reaction should be performed.

Unilateral pulmonary fibrosis rarely needs treatment, for the opposite lung undergoes compensatory emphysema which is usually sufficient for the patient's needs. The only treatment which is likely to be necessary is treatment for underlying tuberculosis or for a complicating bronchiectasis.

(c) *Diffuse bilateral fibrosis* is common in phthisis and it may follow bronchopneumonia. Another common cause is pneumoconiosis, which is a fibrosis of the lungs resulting from the inhalation of dust, and is nearly always an occupational disease. An extreme degree of pulmonary fibrosis may follow inexpert radiotherapy, but with proper technique there is now little risk of this.

The condition is almost always associated with emphysema and the main symptom is *shortness of breath*. As both lungs are equally affected there are few physical signs, but the diagnosis should be suspected when there is increasing *dyspnoea* with a history of one of the causes of diffuse fibrosis. The X-ray shows diffuse strands of fibrous tissue in both lungs. In advanced cases the pulmonary artery and the right side of the heart may be enlarged.

Treatment is symptomatic only.

## FIBROSIS OF THE LUNG

### Pneumoconiosis

281

Fibrosis of the lungs is a common result of the inhalation of certain types of inorganic dust, and it may therefore be an occupational disease. A great variety of conditions is described, according to the type of dust inhaled, but these are all examples of silicosis.

*Silicosis.*—The definition of this condition is important in view of its connection with certain occupations, and that which is the most suitable in practice is "a pathological condition of the lungs due to the inhalation of silica, whether free or combined, in such a state as to be capable of setting up characteristic pathogenic effects" (Lyle Cummins).

The mechanism of production of silicosis is being extensively studied in view of the fact that it is an industrial disease, and a common cause of disablement in districts such as the South Wales coalfield. Certain facts are fairly well established, but additions to our knowledge are to be expected and this account is therefore far from complete.

It appears that it is the small silica (silicon dioxide) particles, from 1 to 3  $\mu$  in diameter, which cause the damage and that they do so because they are to some extent soluble in the tissue fluids. The particles are taken up by phagocytes (koniophages) and deposited on the pathway between the bronchioles and the hilar glands. The root glands are packed with dust, there is a heavy storage of it along the interstitial tracts, in the perivascular and peribronchial sheaths as well as in the interlobular septa and subpleural tissues of the lung. Carbon particles are also present, depending in number upon the origin of the dust. The dissolving silica acts on the neighbouring tissues causing cellular activity, fibrosis and, in the course of time, the formation of fibrous nodules. The process is accentuated by a high alkali content of the dust and is inhibited when aluminium is present. The changes in the lung are complicated by the fact that tuberculosis is frequently present at the same time, and it is difficult in most cases to be sure how much of the change is due to silicosis and how much to infection.

In addition to the fibrosis there is a constant chronic bronchitis, progressive emphysema, and often a mild tubular bronchiectasis. When silicosis has become established it is usual for these lesions to progress gradually. Diffuse fibrosis

and nodule formation are usually present together, but in varying proportions. The bronchi are thickened and possibly dilated, there is emphysema and terminal congestion. In addition, caseation and cavitation occur commonly as a result of tuberculous infection.

The symptoms are those of chronic bronchitis and emphysema, irritating cough, *dyspnoea* which is out of proportion to the physical signs in the chest, and sputum which is said sometimes to be gritty.

The physical signs vary greatly, but in the main they are those of chronic bronchitis and emphysema. As the disease is usually bilateral and symmetrical there is no displacement of the mediastinum. Physical signs of tubercle are often masked by the emphysema. Clubbing of the fingers is common.

The rate of progress of the disease varies directly with the concentration of silica to which the patient has been exposed. The adoption of mechanical rock-drills in the anthracite mines has greatly accelerated the rate of progress of the disease amongst miners. But average exposure must be continued for ten years or more before disabling symptoms make their appearance. Death occurs commonly from tuberculosis, from bronchopneumonia, or from emphysema and right heart-failure.

The diagnosis of the advanced case is not as a rule difficult. Cough and dyspnoea, in a patient whose occupation brings him into contact with silica-containing dust, should always be regarded with suspicion. The X-ray changes are described as (a) reticulation, (b) nodulation, (c) coalescent nodulation, (d) massive shadows, (e) multiple fluffy shadows (figs. 45, 46). The lungs are seen to be uniformly affected, with the exception of the extreme bases. The changes of tuberculosis are often superimposed.

Treatment of the established condition is largely symptomatic. If the patient can live in a healthy, dust-free atmosphere his life may be prolonged for many years. Little can be done for the shortness of breath, but the cough can be relieved by a simple sedative. The treatment is really that of chronic bronchitis and emphysema.

More important is the question of prophylaxis. It is possible in most cases to avoid dust by wearing suitable masks, by damping the dust, or by efficient ventilation systems.

*Asbestosis.*—Asbestos is a fibrous mineral composed of magnesium silicate. The clinical picture is very similar to that of silicosis, but there is not such a close relationship to tubercle. The condition affects those who have worked in contact with asbestos for a period of two years and over, but the clinical state may not become apparent for many years after the occupation has been abandoned.

The lower lobes are chiefly affected by diffuse fibrosis. The underlying pleura is thickened and adherent. There are concurrent bronchitis, emphysema, and bronchiectasis.

Asbestosis pursues the same course as silicosis and the prognosis is always very bad, death occurring from failure of the right heart.

The diagnosis is made by microscopic examination of the sputum, which is found to contain typical "asbestos bodies". These are short and cylindrical, or dumb-bell shaped, and they are bright golden yellow in colour. They consist of deposits of asbestos fibre and the colour is due to an iron-containing pigment, probably derived from haemoglobin. There is no treatment, apart from that of the bronchitis and emphysema, and the course is progressive and inevitable.

*Anthracosis.*—The classical description of this disease is that of a relatively benign condition in coal miners which is due to the deposit of carbon particles in the lungs and glands, with the expectoration of black sputum. It is also said that health is not much impaired and that there is little tendency to the development of tuberculosis.

The above description may be applied to a certain group of cases, but many miners, said to be suffering from "anthracosis", have been shown to be suffering from early silicosis, often with tuberculosis as well. To avoid confusion it has been proposed that the term "dust-reticulation" should be substituted for anthracosis. From the point of view of compensation it has been suggested that the phrase "pneumoconiosis of coal workers" should be used to cover all fibrotic pulmonary conditions due to dust in workers engaged in any operation underground in coal mines, in screens workers at collieries, and coal trimmers at docks.

Certain other occupational affections of the lungs are described. These give rise to clinical pictures of varying degrees of severity. The most common are—

*Siderosis*, which is said to occur in knife-grinders, and to be due to the inhalation of metallic particles. Most of these are in reality cases of silicosis.

weeks. The patient may be put to bed for a few days, but further treatment is not necessary. Should there be evidence of pulmonary tuberculosis, the condition should be treated as such from the start, and it is usually desirable to keep up the pneumothorax by giving refills of air, converting the condition into an artificial pneumothorax. Occasionally a bilateral pneumothorax occurs. This is, of course, a much more dangerous condition and it may be necessary to draw off air from one or both sides.

Cases are on record in which spontaneous pneumothorax has occurred at least ten times with complete recovery in each instance. It follows that the re-expansion of the lung after a simple pneumothorax does not invariably lead to the production of pleural adhesions. In cases of this type treatment may have to be directed to producing pleural adhesions in order to prevent further attacks. The simplest method is to inject from 0.5 to 1 c.c. of a 10 per cent solution of silver nitrate into the pleural cavity while the pneumothorax is still present; this produces a brisk reaction and dense adhesions are quickly formed.

Treatment of the valvular type is a matter of urgency, as the patient becomes increasingly short of breath and is acutely ill. It is necessary to insert a trocar through an intercostal space into the pleura; air escapes in a gush, with instant relief of the symptoms. Unfortunately the air may re-accumulate very rapidly, and it is often necessary to leave the trocar in position and to attach to it a rubber tube which hangs into a bowl of water at the bedside; the excess of air escapes through the tube and the water effectively seals the pleura from the atmosphere. When the opening has healed the lung will gradually re-expand; otherwise a fatal result is likely.

Pneumothorax which is due to a small perforation which remains open does not cause urgent symptoms, for the pressure is atmospheric. The lung will not re-expand and the condition may persist, if untreated, for months or even for years. Pleural symphysis may be obtained by the intrapleural injection of 10 per cent silver nitrate solution.

*Hydropneumothorax.*—A combination of air and fluid in the pleural cavity is not uncommon. If the fluid appears shortly after a spontaneous pneumothorax has occurred the cause of the condition is likely to be tubercle, for spontaneous pneumothorax which results from the rupture of a bulla or cyst is rarely complicated by fluid. A very common cause of hydro-

pneumothorax is a pleural reaction during the course of an artificial pneumothorax. Sometimes hydropneumothorax may be artificially produced by the replacement of fluid by air in cases of pleural effusion. The only symptom which is characteristic of this condition is a splash on movement which may be appreciable by the patient. The other symptoms may be those of pneumothorax or of effusion, i.e. shortness of breath, and perhaps pain, or a feeling of distension in the chest, but there may be no symptoms apart from the splash.

On physical examination the signs will be those of pneumothorax or of effusion, but there is usually a combination.

There is distension and immobility of the affected side and the mediastinum is usually displaced away from that side. The percussion note varies. As a rule there is resonance over the upper part of the chest and dullness below, but the proportion between the two varies according to the relative amount of air and fluid.

The breath sounds may be entirely absent, or there may be amphoric breathing and metallic tinkle. The only pathognomonic sign is *succussion splash* (p. 50). The diagnosis can be confirmed on the X-ray, which shows a *fluid level* (fig. 49).

The treatment of hydropneumothorax is the treatment of the cause. As a rule it is better not to interfere with the fluid, especially in cases of tuberculosis, unless there are pressure symptoms or unless the mediastinum is considerably displaced. In some cases the air becomes absorbed and leaves an encysted effusion which compresses the lung and thus acts as effectively as an artificial pneumothorax, without the necessity for refills.

*Pyopneumothorax*.—This term is applied to cases in which there is air and pus in the pleural cavity. This occurs sometimes in cases of tubercle in which a spontaneous pneumothorax has occurred, and sometimes in patients who are being treated by artificial pneumothorax. It is also a complication of lung abscess and, rarely, of bronchiectasis and of bronchial carcinoma. In war-time the commonest cause is, of course, penetrating wounds of the chest. It is a not infrequent complication of operation on the lung such as lobectomy and of course a temporary pyopneumothorax always results from the open drainage of an empyema.

The symptoms and signs are those of a collection of air and fluid in the chest, but the patient is usually more toxic than



with a hydropneumothorax, there is often considerable fever, leucocytosis, and, in chronic cases, clubbing of the fingers. Succussion splash is not so apparent with pus as with clear fluid. The diagnosis is established by chest puncture.

The treatment depends entirely upon the cause, duration, and general state of the patient. The cases may be divided into two groups.

1. *Simple pyopneumothorax*.—In this case the cause is usually a penetrating wound or a perforated lung abscess. Drainage is necessary and, in the early stages, an intercostal negative pressure drain is safest until sufficient adhesions have formed to localize the infection as far as possible. A bronchial fistula is very likely to result, and further operation may be necessary in order to close this. The condition is serious and has a high mortality. In cases of fistula precautions should be taken to exclude the possibility of latent bronchial carcinoma.

2. *Tuberculous pyopneumothorax*.

(a) *Simple*.—This is a common complication of an ineffective artificial pneumothorax. It often results when refills are kept up in the presence of adhesions which cannot be successfully divided; in all such cases it is far better to abandon the pneumothorax than to run the risk of this event. Treatment should not be drastic at first. Occasional aspiration and general treatment on sanatorium lines are sufficient to secure healing in some cases, but thoracoplasty is ultimately necessary in most.

Promising results have been reported recently from the use of *para-amino salicylic acid* in the treatment of tuberculous empyema. A solution of the sodium salt is injected into the pleura at intervals and, in some cases at least, remarkably quick healing has been found to occur. The drug is at present under investigation in the treatment of other types of tuberculosis.

(b) *With secondary infection*.—This is perhaps the commoner type of tuberculous pyopneumothorax, and treatment is unsatisfactory. Repeated aspiration may assist the patient to overcome the infection, but sinuses are very apt to occur in the course of time. It is often necessary to insert an intercostal negative pressure drain, but open drainage should be deferred as long as possible. General treatment must always be applied as in any other case of active tuberculosis, and the object should be to encourage the lesion to become as quiescent as possible,

in the hope that thoracoplasty may be successfully performed at a later date.

*Hæmopneumothorax*.—This is a comparatively rare condition which follows injury to the chest wall and adhesion cutting. It is a rare complication of spontaneous pneumothorax. The patient should be kept at complete rest until it is certain that the bleeding has ceased, and the blood should then be aspirated as completely as possible, using full aseptic precautions. The blood which is not removed absorbs, leaving an area of gross thickening of the pleura which sometimes calcifies. The condition may become secondarily infected, in which case the clinical state and treatment are those of pyopneumothorax.

there are *no added sounds* when there is a large collection of fluid. It must be remembered that fluid is a good conductor of sound and that the absence of breath sounds is due to collapse of the lung and closure of the bronchi. When there is a small amount of fluid, and part of the lung is collapsed but the bronchus is still patent, *bronchial breathing* will be heard; there may be an area of pleural friction at the upper level of the effusion. The voice sounds are absent over a thick layer of fluid, but at the upper part, where it is less dense, there may be *aegophony*.

The physical signs are sufficiently characteristic to enable the diagnosis to be made correctly in most cases. The distinction from consolidation is sometimes difficult, but the shape of the dullness and the displacement of the mediastinum are significant signs; the vocal vibrations are absent over fluid, whereas they are often increased over an area of consolidation. Effusion and collapse of the lung are easily distinguished by the displacement of the mediastinum, for, with effusion, displacement is *away* from the lesion, with collapse it is *towards* the lesion.

Sometimes effusion may occur with consolidation or collapse. In such cases the displacement of the mediastinum may be misleading. *Whenever there is any doubt about the presence of fluid the chest should be explored with a needle.*

### *Investigations*

1. In every case the sputum should be examined for tubercle.
2. *The pleural fluid.*—About 20 c.cs. should be removed and half of it should be mixed with citrate to prevent clotting.

The typical tuberculous effusion is an exudate which tends to clot spontaneously, the protein content is over 1 per cent, and there are many lymphocytes; varying quantities of blood may be present. It is usually difficult to demonstrate the organisms, but a guinea-pig should be inoculated as a routine, and a positive result is frequently obtained by this means. Cultures may be made on Lowenstein's medium (p. 83).

In cases of malignant growth the fluid may be very similar, but endothelial cells usually outnumber the lymphocytes. Foulis' cells (p. 87) may be found.

3. *An X-ray examination of the chest* is not likely to be helpful when there is a large collection of fluid, for there will simply be a complete opacity on the affected side and displacement of the mediastinum towards the sound side (fig. 11). It is therefore impossible to determine any lung lesion which may be present. With a moderate effusion the opacity is situated in the lower half of the chest and it shows a concave inner margin (fig. 47). In this case the

state of the upper lobe may be studied and tuberculosis or growth may be detected. When it is desired to obtain more information by X-rays the fluid, or part of it, may be removed and replaced by air.

4. In cases of tuberculosis no further investigations are usually necessary, but in cases of suspected growth it may be desirable to do a bronchoscopy.

5. Finally, should the cause still be doubtful, a thoracoscope may be inserted after an air replacement has been done, and the pleura may be directly inspected. It is even possible to remove tissue by this means for histological examination.

The treatment of pleural effusion depends entirely upon the cause. When it is considered to be due to tuberculosis absolute rest in bed is necessary while the patient is febrile, and for at least two weeks after the temperature has returned to normal. Unless the effusion is sufficiently large to cause pressure symptoms it is better not to withdraw any large quantity of fluid for at least six weeks. During this interval it will be found that, in many cases, absorption begins and may progress quite rapidly, so that paracentesis is unnecessary. Should the fluid show no sign of diminishing after six weeks a quantity up to two pints may be removed. In most cases it is sufficient simply to remove the fluid, and this procedure is often followed by rapid absorption. When there is a tendency for the fluid to re-accumulate, or when there is any doubt as to the underlying cause, an air replacement should be done.

When the fluid has practically absorbed, and the temperature has been normal for two weeks, the patient may be allowed to get up and slowly resume a life of moderate activity. It is debatable whether sanatorium treatment should be advised, but it may be stated that, when there is evidence of active lung disease, the patient should be sent to a sanatorium, and should have the fluid replaced with air, in order to produce an artificial pneumothorax. When the effusion is primarily due to disease of the pleura there is a danger that a further infection, such as may easily occur in a sanatorium, might cause a flare-up of a latent lung lesion. In such cases it is better to send the patient for a long and careful convalescence by the sea, under medical supervision.

The patient's life should be carefully regulated and the return to normal activities should be very gradual. Sun bathing should be absolutely prohibited, and sea bathing should

be discouraged for at least three years after the effusion has completely absorbed.

It is a wise plan to overhaul the patient, and to take X-rays, at increasing intervals. Subsequently any sputum which is coughed up should be examined as a precaution.

*Encysted effusion.*—Sometimes a pleural effusion occupies a part only of the pleural cavity, being shut off by adhesions. The physical signs may be very difficult to interpret. As a rule the diagnosis is only made for certain after exploration with a needle. This should only be done after careful examination of postero-anterior and lateral films of the chest. Fig. 48, which shows an oleothorax, represents what is, in effect, an encysted effusion.

*Interlobar effusion* is uncommon and the diagnosis is difficult. The physical signs may be very few, and the condition is often first suspected after inspection of postero-anterior and lateral X-rays. A diagnostic puncture is usually desirable but, when the fluid is sterile, it is best left untouched. Fluid which becomes encysted in this situation may remain for many years without giving rise to any trouble.

*Haemorrhagic effusions* are nearly always tuberculous or malignant. The clinical picture is that of a simple effusion and the diagnosis is only made on exploratory puncture; the treatment is that of the cause.

*Haemothorax* implies the presence of pure blood in the pleural cavity. The blood most commonly comes from the parietal pleura and usually results from chest injury. Spontaneous haemorrhage is sometimes seen, usually when the lung is adherent to the chest wall, and rupture of an aneurysm is a rare cause. The great danger, especially in the traumatic cases, is secondary infection. An uninfected haemothorax should be aspirated as completely as possible between the second and fourth days, and an air replacement may be done at the same time; a further aspiration may be necessary a few days later. The lung often expands completely. In most cases the blood remains liquid and there is no difficulty about aspiration. Sometimes a clot is formed and this should be removed if it is of considerable size. An infected haemothorax is treated by closed intercostal drainage under a water seal. Foreign bodies in the pleura may be left until later; those in the lung should be left altogether unless they are causing symptoms.

*Chylous pleural effusion.*—In these cases the diagnosis can only be made by pathological examination of the fluid. When the fluid is pseudo-chylous, resulting from nephritis or from some other general cause, the treatment is that of the cause. When pseudo-chylous fluid is found in an encysted effusion of long standing, and probably tuberculous, it is best left alone.

The presence of true chylous fluid in the pleural cavity indicates damage to the thoracic duct, damage which is often irreparable.

*Paracentesis of the pleura.*—Both for diagnosis and for treatment it is often necessary to insert a needle into the pleural cavity. The procedure is simple when properly carried out and it should be entirely painless, for a local anaesthetic must always be used. On the Continent the chest is often explored, or a refill given, without any anaesthetic but, although this procedure is less painful than might be expected, there is a distinct risk of pleural shock with collapse and, occasionally, sudden death. For this reason alone it is wiser to be on the safe side and never to puncture a sensitive pleura. The indications are as follow :—

- (a) To remove fluid for examination.
- (b) For the aspiration of an effusion.
- (c) When there is uncertainty as to the presence of fluid.

The site of the puncture must first be selected. In most cases, when the effusion is situated in the main pleural cavity, it is best to enter the chest one or two spaces below the angle of the scapula. The spaces in this region are reasonably large, and the patient cannot see what is being done. Should there be reason to suspect that the fluid is situated in the interlobar space it is better to explore in the 5th or 6th space in the mid-axillary line for, from this situation, it is possible to investigate a large area of the chest by varying the direction of the needle. When the fluid is encysted the site of exploration should be selected after a careful examination of postero-anterior and lateral X-ray films.

The selected site is cleaned and the direction of the intercostal space is found by placing the index and middle fingers of the left hand in the space about one inch apart, stretching the skin slightly between the fingers. By doing this the exact area to be punctured is clearly defined and the possibility of striking a rib is eliminated. A 2-c.cs. syringe is filled with 2 per cent novocaine solution and an intradermal needle is attached. About 0.2 c.c. of the solution is then injected into the skin immediately over the space. This raises a small wheal which can be easily identified. An intravenous needle is then fixed

to the syringe and pushed through the wheal. The subcutaneous and intramuscular tissues are practically insensitive, so that it is not necessary to inject any more novocaine until the parietal pleura is reached. At this point the patient will experience a slight pricking sensation, and about 1 c.c. of novocaine is then injected. The needle is pushed on and more novocaine is injected until it is certain that the pleura is properly anaesthetized. It is usually possible to confirm that the pleura has been penetrated by applying suction to the piston of the syringe for, when the pleura is passed, fluid, air, or froth will be withdrawn. The needle used to reach the pleura must always be a little longer than the distance to be travelled, so that it is never necessary to insert it up to the hilt; if the needle should happen to break it can be readily removed with forceps.

A wide-bore needle is next attached to a 20-c.cs. syringe and passed into the pleural cavity. Ordinary pleural fluid can be withdrawn quite easily but thick pus is often difficult to remove. If nothing can be obtained in the syringe it is as well to note the contents of the needle itself, as a little pus may be detected in its lumen.

*Aspiration of the chest.*—When fluid has been found it is sometimes necessary to remove it. The indications for aspiration are—

(a) *Tuberculous pleural effusion* which has not shown signs of absorption after six weeks.

(b) *Rapidly increasing effusions* which are causing dyspnoea.

(c) Symptoms of intrathoracic pressure in cases of growth.

(d) Empyema containing thin pus, usually streptococcal.

The amount of fluid to be removed varies in different cases but as a rule not more than two pints should be removed at one time. Should it be necessary to remove a larger amount it is advisable to replace the fluid with half the quantity of air. The patient sometimes complains of pain and develops an irritative cough, with watery sputum and streaks of blood, during the course of an aspiration. This indicates that the lung is expanding too rapidly, and the aspiration should be stopped; the admission of a small quantity of air usually relieves the symptoms at once.

Aspiration may be done either with an aspirating bottle or with a special syringe.

(a) *Aspiration by means of a bottle.*—A suitable apparatus has been designed by the author. It consists of a flat-bottomed bottle of about 300 c.cs. capacity with two apertures, the upper of which is closed by a rubber stopper which carries a glass tube of 11 mm.

diameter. The outlet, which is situated in the side of the bottle about 1 cm. above the base, is closed by a rubber stopper which carries a glass tube guarded by a stop-cock. The upper glass tube is connected by means of rubber tubing with an aspirating needle, and tubing leads from the outlet to a vessel on the floor. The whole apparatus is filled with water and it is then ready for use. The flow is started simply by opening the stop-cock, which establishes syphonage, and the rate of flow can be readily controlled by adjusting the tap. The apparatus possesses certain advantages. It is quite stable and can be rested on any convenient flat surface, such as a chair, thus avoiding the necessity for attachment to a bed-rail, which is not always convenient; the stop-cock takes the place of the customary clip and is much simpler to operate, so that the rate of flow can be readily adjusted. It will be noted that the exit tube is situated a short distance above the bottom of the bottle, thus allowing for the accumulation of debris and minimizing the chance of blocking the outlet. The apparatus is readily portable.

This bottle can be used for the aspiration of all types of fluid from the chest, even pus of medium consistency; occasionally, when there is much fibrin in the fluid, clotting may occur in the stop-cock, but the clot can be easily dislodged. When there is reason to anticipate trouble with clotting the bottle can be filled with citrate solution at the start. It is essential that both stoppers should fit tightly in order to prevent the entrance of air into the apparatus.

(b) *Aspiration by means of a syringe.*—Many special syringes have been devised for this purpose, most of which have two or three nozzles. A suitable type is the "Rotanda", which has a rotating barrel. The rubber tubing attached to one of the nozzles carries the needle which is inserted into the chest; withdrawal of the piston removes the fluid. The barrel is then rotated and the fluid can be expelled through another outlet into the collecting vessel. The third aperture can be used for the admission of air or antiseptic fluid into the chest if desired.

*Air replacement.*—It is sometimes necessary, after the removal of fluid, to leave a quantity of air in the pleural cavity. This is done to relieve symptoms when fluid has been removed too rapidly, to slow down the rate of accumulation in recurrent effusions, and, sometimes, to enable further X-rays to be taken in order to see the state of the lung and the pleura. The simplest method is to remove the rubber tubing from the needle, holding a small piece of sterile gauze over the end; when there is a negative pressure air will be sucked in until the pressure is atmospheric. When an aspiration is being done with a three-way syringe, air can be pumped in through the same needle but, when setting out to do an air replacement, it is better to make



two punctures in the chest, one for the removal of the fluid and the other for the introduction of air. The fluid is removed through the puncture in the lower part of the chest and the needle for the introduction of air is inserted at some convenient spot, about the upper limit of the effusion. The position selected is usually in the second intercostal space in front, or high up in the axilla. Fluid is first withdrawn until it ceases to flow freely, and the second needle is then connected to a pneumothorax apparatus and inserted into the pleura. It is better to let the air be drawn in by the suction caused by the removal of the fluid, rather than to attempt to force air into the chest at a positive pressure.

## CHAPTER 33

### EMPHYEMA

THE term "empyema" indicates the presence of pus in the pleural cavity. The conditions in which it most commonly occurs are—

1. *Lung Infections.*
  - (a) Lobar pneumonia and bronchopneumonia.
  - (b) Lung abscess.
  - (c) Bronchiectasis.
  - (d) New growth.
2. *Blood Stream Infections.*

As a complication of septicaemia and pyaemia.
3. *Wounds of the Chest.*
  - (a) Gun-shot wounds and other perforating injuries
  - (b) Following aspiration; during pneumothorax treatment.
4. *Lesions of the Mediastinum*

Perforation of the oesophagus by foreign body or carcinoma.  
Spread of suppurative mediastinitis.
5. Upward extension from abdominal infection.
6. *Tuberculous empyema.*

Empyema is commonly due to the pneumococcus or to the streptococcus. Occasionally, especially in pyaemia, the staphylococcus is found and, in some cases of lung abscess and growth, there may be anaerobic organisms. The other common bacteria, *M. catarrhalis*, *H. influenzae*, and *B. Friedlander*, are sometimes found. Finally, in all cases of chronic empyema, the possibility of tuberculosis and actinomycosis should be considered.

The symptomatology of empyema is not clearly defined. In the cases which follow lobar pneumonia there is an imperfect recovery from the original infection. In other cases there may be the symptoms of a lung infection, with cough and sputum.

*Toxaemic symptoms.*—Malaise, anorexia, dyspnoea out of proportion to the physical signs, rigors, sweats, and loss of weight.

*Symptoms of the local lesion.*—There may be pain in the early stages but this usually passes off, and there may be little in

the subacute and chronic stages to indicate disease in the chest. Cough and sputum are the result of the lung infection. The temperature is usually hectic, being raised at night and often normal in the morning.

Pathologically there are three chief types of acute empyema.

1. *Pneumococcal empyema* is characterized by a copious exudate of fibrin which tends to organize and to shut off the empyema from the surrounding pleural cavity. A pneumococcal empyema is therefore suitable for open drainage.

2. *Streptococcal empyema* causes a copious effusion of thin pus and as a rule the whole pleural cavity is involved (pyothorax). In this type there is little tendency to the formation of adhesions and the mediastinum is frequently mobile. It is important to bear this in mind when considering an operation for open drainage.

3. *Staphylococcal empyema* is secondary to a lung abscess or to pyaemia. It is always associated with intense toxæmia and until recently it has ranked as the most dangerous type.

It was hitherto customary to distinguish between an empyema which arose before the crisis (synpneumonic empyema), and one which developed after the crisis was passed (metapneumonic empyema), but the introduction of modern methods of treatment has deprived this distinction of most of its point. Chemotherapy now controls the infection, and the temperature, so speedily that the diagnosis is made on the physical signs and not on the temperature chart. It has been suggested that empyema is commoner in cases treated by sulphonamide, but this is doubtful.

The physical signs are similar to those of any other collection of fluid in the pleural cavity but, in the type with much fibrinous exudate and consequent adhesions, there may be little displacement of the heart. Sometimes an empyema may be overlooked for weeks and, in such cases, clubbing of the fingers may develop. Persistent toxæmia is always suggestive of the presence of pus in the chest. The X-ray does not distinguish pus from any other type of fluid, and the diagnosis is established by chest puncture; a wide-bore needle must always be used when the presence of pus is suspected. Localized empyema may be indistinguishable from a lung abscess without an exploratory operation.

*Treatment.*—The treatment of acute empyema has been greatly modified since the introduction of penicillin, for the common causal organisms, the pneumococcus, streptococcus, and staphylococcus, are usually sensitive. It is not possible as yet to lay down any rules for universal application, but the following principles may be taken as a guide.

(a) *Turbid effusions*, while the patient is still toxæmic. The fluid is aspirated as completely as possible, and 240,000 units of penicillin in 20 c.cs. of saline are injected slowly into the cavity. This procedure is repeated every second day until fluid ceases to form or, as happens more frequently, until the effusion is frankly purulent. Treatment is then continued as is outlined in (b).

(b) *Purulent effusions* are best treated by *closed intercostal drainage*. An incision is made in the skin and a wide-bore trocar and cannula are pushed through the parietal pleura; the trocar is removed and a self-retaining catheter is introduced into the empyema, the cannula being slipped out over the tube. The catheter is then connected to a bottle containing water in order to form a seal, and the pus is allowed to drain by gravity. The tube must be placed low enough to secure full drainage, yet not so low as to be obstructed by the diaphragm as it returns to meet the chest wall.

Instillations of 60,000 units of penicillin are given through the tube twice daily and the end is closed with a spigot for eleven hours out of twelve; during the remaining hour drainage is quite sufficient to keep the cavity empty.

Treatment is continued until pus ceases to be formed and the discharge is serous and scanty. At this stage the tube can be removed and a dry dressing applied. A pleurogram (p. 68) should be done before the tube is removed in order to be sure that the cavity is closed.

(c) *Thick pus*, especially if accompanied by massive fibrin formation, should be drained by rib resection. This is most likely to occur with pneumococcal infection. In these cases a full course of systemic penicillin is likely to have been given during the active stage of the pneumonia and there is little to be gained by attempting to instil penicillin into the open cavity.

(d) *Staphylococcal empyema* is always a dangerous condition, and is best treated by full doses of intramuscular penicillin, together

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When there is a fistula a local operation may be successful in repairing the damage but thoracoplasty is often necessary.

In all cases of chronic empyema the usual investigations must be undertaken to exclude tuberculosis, actinomycosis, and growth.

*Tuberculous empyema.*—For practical purposes this may be divided into two groups, according to whether secondary infection is present.

(i) Tuberculous empyema which is not secondarily infected may be treated by very careful repeated aspiration, combined with the intrapleural injection of a solution of azochloramide, 1 in 2000, with sodium tetradecyl sulph., 1 in 800; the injections are given daily for three days and then twice weekly for about three months. The results of this method are good enough to render it the treatment of choice in this type of case. Failing this, an intercostal drainage under a water-seal may be established with the object of inducing re-expansion of the lung and obliteration of the pleural space. In some cases this may be sufficient but, if there be evidence of persistently active disease in the lung, a thoracoplasty should be done without undue delay.

The use of the sodium salt of *para-amino salicylic acid* has already been noted (p. 270). It is not yet possible to describe this as the treatment of choice but, if the preliminary results are confirmed, it seems likely that it will supplant the other methods in the near future.

(ii) A secondarily infected tuberculous empyema may result from the rupture of a superficial cavity, but it is most commonly seen in cases of pneumothorax treatment complicated by adhesions. It is much safer to abandon such a pneumothorax if the adhesions cannot be divided. Closed intercostal drainage with penicillin instillations will usually suffice to quell the pyogenic infection and treatment is then carried out as for a tuberculous empyema. In these cases thoracoplasty is almost inevitable.

*Broncho-pleuro-cutaneous fistula.*—A common cause of chronic empyema is an opening between the pleural cavity and the bronchi. This occurs in cases of perforated lung abscess, growth, or injury to the lung from wounds or operations. Sometimes the presence of the fistula can be readily recognized by a hissing noise produced when the patient breathes deeply

with closed intercostal drainage and penicillin instillations. The mortality has been much reduced by penicillin treatment.

If an empyema is not recognized various things may happen.

1. Absorption of the pus may occur, leaving a grossly thickened pleura or even a calcified area. The lung may remain collapsed and fibrosis may follow.

2. The empyema may present and burst through the chest wall, usually anteriorly (*empyema necessitatis*). In this event there may be a persistent sinus, but the patient often does well. Superficially this condition might resemble an aneurysm.

3. The empyema may rupture into the lung and may be coughed up. Usually this produces a *pyopneumothorax* which needs surgical treatment, but spontaneous recovery sometimes occurs.

4. The empyema may remain latent for years. The patient suffers from the symptoms of general toxæmia, clubbing is often intense, and amyloid disease may develop.

Complications of empyema are not very common. Spread of the infection may result in pericarditis, or there may be a septicaemia or pyaemia and, in some cases, *brain abscess*.

*Chronic empyema* may occur in the following ways:—

1. When an acute empyema has been overlooked and drainage has eventually been carried out after the walls of the cavity have become too thick to permit easy expansion of the lung.

2. Following inadequate drainage. The opening into the cavity should always be situated near its most dependent part.

3. When there is a broncho-pleural fistula.

4. With a foreign body.

5. With tuberculosis and actinomycosis.

6. With an underlying abscess or growth.

Post-operative chronic empyema results from failure of expansion of the lung, either from gross pleural thickening or from fistula formation. In the first group various methods of remedial treatment may be tried.

(a) Breathing exercises designed to encourage expansion of the affected lung. These are not usually very successful.

(b) An operation for decortication; the thickened membrane is stripped from the surface of the lung. Re-expansion may follow this procedure.

(c) Thoracoplasty may sometimes be necessary to close the cavity.

## CHAPTER 34

### TUMOURS OF THE PLEURA

Tumours in connection with the pleura are uncommon. They may be innocent or malignant. Innocent tumours may be found in the visceral or parietal pleura, the least rare being lipoma, fibroma, and neuro-fibroma. These tumours may give rise to very little in the way of symptoms or signs, but pain is perhaps the chief symptom; cough, sputum, and haemoptysis do not occur with innocent tumours. The diagnosis is made on the X-ray, which shows a well-defined round or oval opacity. It is often difficult to be sure that the tumour is really in the pleura, and not in the lung itself, and in such cases it is necessary to induce a pneumothorax and to take further X-rays; the lung can then be seen to have moved away from the opacity. These tumours are often removable. They must be distinguished from gumma, or from aneurysm of the descending aorta; the Wassermann reaction should always be done and, in cases of doubt, an exploratory operation may be justifiable. So long as the operation is carefully carried out, no harm is likely to result.

Primary malignant tumour of the pleura is nearly always an endothelioma, the malignancy of which varies a great deal. The symptoms are pain and shortness of breath. Pain is due to nerve involvement and dyspnoea to pleural effusion, which may accumulate very rapidly. In other cases there is a large mass of solid tissue which obliterates the pleural space, and which has the histological characters of a fibrosarcoma. In cases with pleural effusion an air replacement may permit a thoracoscopy to be performed in order to establish the diagnosis. Some of these tumours are sensitive to X-rays but as a rule they are so extensive that treatment cannot be effectively carried out. It only remains to be added that many of the cases which have been described as primary malignant disease of the pleura are in reality examples of the pleural spread of an underlying bronchial carcinoma.

Secondary involvement of the pleura may occur by direct spread from a carcinoma of a bronchus or breast, or as a result of metastasis from a malignant tumour anywhere in the body. Treatment in these cases is not at present possible.

or coughs. At other times there may be no special symptoms or signs, and in these cases it is necessary to introduce lipiodol through the opening in the chest wall into the pleural cavity ; when a fistula is present the oil is seen to have entered the bronchi (fig. 51). It is necessary to examine the sinus in the chest wall before doing an ordinary bronchogram, otherwise the result will be inconclusive.

Sometimes a fistula closes spontaneously but at other times a local operation and cauterization are necessary ; in very chronic cases thoracoplasty may have to be done.

## CHAPTER 34 TUMOURS OF THE PLEURA

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*Lymphadenoma.*—Enlargement of the mediastinal glands is common in Hodgkin's disease. When the condition is typical, with enlargement of glands in other situations, and with enlargement of the spleen, there need be no doubt about the diagnosis, for a gland can be removed for section. The histological features are : (1) The normal lymphoid structure of the gland is destroyed and is replaced by (2) proliferation of endothelial cells. (3) Some of the endothelial cells enlarge to form typical giant cells, which are not very large and contain three or four centrally situated nuclei. (4) There are usually many eosinophil cells. (5) In the later stages fibroblasts are present and there is an increasing degree of fibrosis.

The symptoms and signs are those of a mediastinal tumour, often with relapsing pyrexia (Pel-Ebstein syndrome). The X-ray shadow (fig. 54) is not sufficiently characteristic to enable the diagnosis to be made in the absence of a microscopic section. Conditions which have to be distinguished are tuberculosis, growth, and leukaemia. Tuberculosis and lymphadenoma often coexist.

Treatment is by deep X-rays. Arsenic is also given, both by the mouth and intravenously. Although the disease can often be controlled for a time the ultimate prognosis is hopeless.

### Mediastinal Tumour

Primary tumours in the mediastinum are not so common as was thought at one time to be the case, for many apparent examples are in reality type 3 bronchial carcinomata. Secondary carcinoma and sarcoma are also found.

The symptoms of them all are essentially similar, for they result from pressure upon the important surrounding structures. The chief complaints are irritating and useless cough, dyspnoea, dysphagia, and pain. The pain may be a dull ache due to pressure, or an acute radiating pain due to involvement of intercostal nerves. Aphonia may occur from involvement of a recurrent laryngeal nerve.

The physical signs will depend upon the size and situation of the mass. On inspection there may be cyanosis, dilatation of superficial veins, and oedema of the face and arms. A swelling is rarely visible on the chest wall, except with aneurysm. The movements are symmetrical as a rule; sometimes there is

asymmetry when the tumour projects to one side. The heart is not likely to be displaced. There is often an area of dullness of varying size, and there may be local weakness of breath sounds, or bronchial breathing. Pleural effusion is present in some cases, usually a transudate.

The X-ray reveals a well-defined mass situated in the centre of the chest which does not pulsate. The shape varies and the lung field may be invaded, or the diaphragm paralysed by a malignant tumour.

Certain special tumours must be mentioned separately.

*Retrosternal pituitary.*—This is a not uncommon tumour and sometimes there is no obvious enlargement of the thyroid gland in the neck, although the patient may show signs of thyrotoxicosis. There is often a sense of local pressure, sometimes taken to be "functional" in origin, and dysphagia. Dyspnoea and stridor occur later. The diagnosis is made on the X-ray which shows a pear-shaped shadow, often with its base resting on the arch of the aorta, which may be displaced. The trachea is either displaced or narrowed (fig. 53). On the screen the shadow is seen to move upwards on swallowing. Most of these tumours are removable.

*Innocent tumours.*—Fibroma, neurofibroma, myoma, and lymphoma have all been described, but they are very rare.

*Malignant tumours.*—Sarcoma, usually a lymphosarcoma, is occasionally seen. It is a rapidly-growing tumour and pressure symptoms and signs occur early; it is usually very radio-sensitive (figs. 57, 58). Masses of secondary carcinoma or sarcoma are commoner. Their recognition depends upon knowledge of the existence of the primary growth.

*Cysts.*—Dermoid cyst and teratoma are both occasionally seen. The dermoid is usually situated in the anterior mediastinum, often in connection with the pericardium, or in the superior mediastinum, arising from thymic remains. It is surprising how few symptoms may be caused by quite a large cyst, but eventually pressure symptoms and signs occur. The X-ray shows a well-defined oval shadow (fig. 52); sometimes the presence of teeth or bone may be demonstrated. A dermoid cyst may become infected, in which case the patient becomes acutely ill; the prognosis is bad with infection. There is a tendency for a teratoma to become malignant in the course of time.



Dermoid cysts should be removed as a rule. If the cyst should be too large to be delivered with ease it may be marsupialized and removed at a subsequent operation.

*Thymic tumours.*—Moderate enlargement of the thymus is sometimes found at post-mortem examination of children who have died suddenly and unexpectedly (*status thymico-lymphaticus*). There are no definite symptoms nor signs of enlargement of the thymus and the X-ray appearances are inconclusive. Occasionally a mild degree of stridor in children has been thought to be caused by the pressure of an enlarged thymus (Kopp's asthma). The gland is found to be enlarged in cases of myasthenia gravis, and it has been removed in such cases with relief of symptoms.

Malignant tumours, carcinoma and sarcoma, are rarely seen. Although the onset of pressure symptoms is almost invariably sudden, the tumour is already quite large when the patient is first seen. The X-ray appearance is that of a rectangular mass in the anterior superior mediastinum. Carcinoma of the thymus is completely radio-resistant.

*"Dumb-bell" tumour.*—This is a rare tumour, situated in the posterior mediastinum and joined by a stalk to a similar, but smaller, mass in the vertebral canal. The chest tumour is usually latent, and the patient complains of symptoms of pressure on the spinal cord. Both parts of the tumour may be removed.

### *Aneurysm of the Aorta*

An aneurysm is a dilatation of an artery, which may be either localized or diffuse. Aortic aneurysm usually results from *syphilis*, although in some cases the weakening of the vessel wall results from atheroma. Apparently the syphilitic infection reaches the wall of the aorta by way of the lymphatics from the mediastinal glands, and the first vessels to be attacked are the nutrient arteries which supply the wall of the aorta. These show the common syphilitic changes, *endarteritis* with consequent narrowing of the lumen, and *periarteritis* with round-celled infiltration, plasma cells, and fibrosis. These changes reduce the nutrient blood supply and result in scarring and weakness of the *middle coat of the aorta at first* (*mesaortitis*). The most marked changes are in the ascending part, and the damage becomes progressively less towards the descending

arch. The inner part of the aorta is seen to be thickened and contracted, with small linear or stellate scars which have a characteristic greyish colour. This appearance is pathognomonic of syphilis. Atheromatous changes may of course coexist. It is rare to find sufficient necrosis present to result in gumma formation although this has been described. There may be associated scarring of the aortic valve, with consequent secondary changes in the left ventricle. Syphilis may give rise to two distinct types of dilatation, *saccular* and *fusiform*. The former is the commoner, and the one which produces the most striking changes. The clinical picture varies according to the site of the aneurysm.

1. *Aneurysm of the ascending arch* ("aneurysm of physical signs").—The most common symptom is *pain* in the right side of the chest which results from erosion of the ribs or sternum, or there may be anginal pain due to involvement of the orifices of the coronary vessels. *Cough* may be due to pressure on the right bronchus and there may be *haemoptysis*.

The physical signs of a large sac are characteristic. There is a swelling in the second right interspace which *pulsates*, and this pulsation is *expansile*. On palpation there may be a diastolic shock, due to the mass of blood falling on the aortic valve, and a systolic thrill can sometimes be felt. It is usually possible to map out the area of dullness on percussion. There may be an accentuated aortic second sound and systolic or diastolic murmur.

Additional signs are those which result from pressure on surrounding structures. There are sometimes signs of collapse of the right upper lobe. Rarely there may be dilatation of the veins over the front of the chest and oedema of the face or right arm. Sometimes the right pupil is larger than the left; it appears that this is not usually due to sympathetic irritation, but to a lower blood pressure in the right carotid artery, with consequent constriction of the vessels of the iris. Sometimes the sympathetic is involved, in which case there will be more marked dilatation of the right pupil, with some degree of exophthalmos and unilateral flushing and sweating. The common termination of this type of aneurysm is by *rupture*, either externally, or into the right pleural cavity, or into the pericardium. Death may also occur from heart-failure.

2. *Aneurysm of the transverse arch* ("aneurysm of sym-

toms").—In view of the confined space through which so many vital structures pass, it follows that a comparatively slight dilatation of the transverse aorta will give rise to very pronounced symptoms. Irritating cough, "brassy" or "bovine", is almost constant; *dyspnoea* and *dysphagia* also occur early. There may be alteration in the voice from pressure on the left recurrent laryngeal nerve.

The physical signs are much less definite than with aneurysm of the ascending arch. In some cases a pulsating swelling may be seen above the jugular notch of the sternum, and there may be pulsation behind the manubrium. The signs of pressure are perhaps more significant, for there may be laryngeal paralysis, inequality of the pupils, and dilatation of the veins with oedema. In addition there are two significant signs which result from the aneurysm itself: (a) *Tracheal tug*. When the neck is slightly extended, gentle upward pressure on the larynx elicits a tugging sensation, systolic in time, which results from the pulsating aneurysm dragging on the bifurcation of the trachea. (b) *Inequality of the pulses*. The presence of the sac results in a lowering of systolic pressure on the left side and a retardation of the flow of the blood. Thus there may be *asynchrony*, the left pulse being slightly later in time than the right, or *lessening of the volume* of the left pulse. The latter fact can be confirmed by comparing the blood pressure on the two sides.

Death may result from heart-failure, from tracheal compression, or from rupture into a bronchus or into either pleural cavity.

3. *Aneurysm of the descending arch*.—In this type there is a diversity of symptoms and signs, but the condition may remain latent for a long time and may cause much difficulty in diagnosis. The sac extends chiefly backwards and to the left, so that vital structures are not likely to be involved early. The chief symptoms are *pain*, which may be very severe and usually results from erosion of the vertebrae and pressure upon the posterior nerve roots; *cough*, which is caused by pressure on the left bronchus; *dysphagia*, from pressure on the oesophagus, which runs in close relation to the descending arch. When there is marked erosion of the bodies of the vertebrae there may be pressure on the spinal cord itself, with *spastic paraplegia*.

The signs are usually those of a tumour and there is

little clinically to suggest aneurysm. There is dullness in the left interscapular space, weak breath sounds, and bronchial breathing. Secondary changes in the left lung, bronchitis, bronchiectasis, and collapse may occur and give rise to their usual physical signs. In some cases there may be a pulsating tumour in the left interscapular space and a systolic murmur may be heard over the sac, but both of these signs are rare. Examination of the nervous system may show evidence of a spinal cord lesion. Death occurs from rupture into the left pleural cavity, or from some lung infection such as pneumonia or abscess.

4. Saccular aneurysm may occur in the descending aorta below the arch. The symptoms are similar to those of aneurysm of the descending arch, but the pain may be referred to the abdomen. The signs are inconclusive as a rule, and the diagnosis must be made on the X-ray screen.

5. Fusiform aneurysm commonly involves the ascending arch, but it may be more extensive, involving the whole of the arch, or even the whole of the aorta. This type is usually due to syphilis (fig. 55), but it may occur with atheroma. The symptoms are cardiac rather than respiratory, and the patient complains of anginal attacks, shortness of breath, and, less commonly, difficulty in breathing. The physical signs are usually not conclusive, although there may be an increase of the mediastinal dullness. The aortic valve is often affected and there may be an aortic diastolic murmur. In this type screening is essential in making the diagnosis. Death usually occurs from coronary disease and heart-failure.

The diagnosis of aneurysm may be very simple or very difficult. A positive Wassermann reaction is to be regarded as being in favour of aneurysm, but a negative reaction does not exclude it. In doubtful cases the appearance on the X-ray screen must be carefully studied. An aneurysm is sharply defined (fig. 56), and the presence of pulsation is important. Occasionally, when there is a massive clot in the sac, pulsation will be absent; rarely a sarcoma may be so vascular that it appears to pulsate. Aneurysm does not cause paralysis of the diaphragm. In doubtful cases the kymogram (p. 69) may confirm the presence of expansile pulsation. Rest has some effect in relieving the symptoms, and even in causing the sac to

diminish in size. Dietetic measures to increase the coagulability of the blood by starvation are valueless, as are such drugs as calcium and gelatine. In syphilitic cases intensive treatment should be given, as the condition may be arrested although it cannot be caused to retrogress. Intravenous arsenical compounds must be given with extreme caution when the lesion is near the mouths of the coronary arteries.

In cases of saccular aneurysm, especially when close to the surface, wire may be introduced into the sac in order to promote clotting and thus to strengthen the wall. In some cases this operation is very successful, and the sac may even diminish considerably in size. There can be no doubt that, in carefully selected cases, the operation may be responsible for considerable prolongation of life.

## CHAPTER 36

### THE DIAPHRAGM

The diaphragm forms an important part of the respiratory mechanism and it has already been considered in relation to various chest conditions. There are some diseases in which the muscle is primarily at fault.

*Diaphragmatic hernia.*—The abdominal contents may find their way into the chest through a gap in the muscle. This may result either from congenital absence of a part of the diaphragm or from some cause such as injury. The lesion is more common on the left side than on the right. As a rule the stomach and, less commonly, the colon are found in the left pleural cavity, separated from the pleura by a thin layer of fibrous tissue. The size of the sac varies greatly in different cases. When the lesion is on the right side the liver tends to block the opening and to prevent the escape of the other abdominal contents, although sometimes the colon may be found in the right pleural cavity (figs. 61, 62).

The symptoms are very variable and there may be none for many years. In fact, the initial symptoms often do not manifest themselves until middle age. There may be symptoms of pressure in the pleural cavity, such as dyspnoea, or there may be dyspepsia, vomiting, and flatulence.

The physical signs are very varied and they may simulate pneumothorax, effusion, or consolidation. A significant feature is rapid alteration in the signs. There may be gurgling noises, such as are commonly heard over the abdomen, and there may even be the appearance of a void in the hypochondrium on the affected side. The diagnosis is usually made on the X-ray, although a plain film may be misleading, for it may suggest pneumonia or tumour. In all suspicious cases a barium meal or enema should be given, after which there can be no further doubt as to the diagnosis.

Patients in whom the condition is discovered accidentally, and who are not complaining of any symptoms, are best left alone. When symptoms demand relief an operation should be

attempted. Usually a combined operation is done, the chest and abdomen both being opened, and the abdominal contents being restored to their proper place. The rent in the diaphragm is then repaired and, if necessary, a graft can be added to strengthen the muscle; this is usually necessary in the congenital cases, where the tissue is more fibrous than muscular. The results of a successful operation are excellent.

*Eventration of the diaphragm.*—This is a congenital condition in which the muscle fails to develop, and the pleura and peritoneum are separated by a thin layer of fibrous tissue. It appears to occur only on the left side and almost always in males. The condition does not give rise to any special symptoms, and such physical signs as are present are those of a high diaphragm. The diagnosis is usually made on the X-ray (fig. 6). Treatment is not necessary.

*Paralysis of the diaphragm.*—This is most commonly nowadays the result of a phrenic avulsion, but it may occur in diphtheria, in poliomyelitis, or as a result of damage to the nerve by wounds, or by injury to the spine in the region of the third and fourth cervical segments. It frequently results from infiltration of the nerve by malignant growth in the chest (fig. 30), but not from aneurysm.

There are not usually any definite symptoms of paralysis of one side of the diaphragm; when both sides are involved in cases of polyneuritis or poliomyelitis there may be such respiratory embarrassment that the patient may die from respiratory failure.

The physical signs of unilateral paralysis are indefinite, but the affected side of the chest may be seen to move rather less than the normal and there may be a distinct void in the hypochondrium. There may also be impairment of percussion note and weakening of the breath sounds at the base of the corresponding lung. There will, of course, be an absence of inspiratory recession of the intercostal spaces in the mid-axillary line (Litten's sign).

The state of the diaphragm is best appreciated on the X-ray screen, for it can be seen to be immobile, or even to exhibit *paradoxical movement* (p. 52). On the film the paralysed diaphragm is seen to be raised (fig. 30).

*Hiccough.*—This symptom arises from an irritation of the diaphragm as a rule. It may result from the following causes:—

(a) Most commonly from dyspepsia or from taking irritating substances such as hot liquids, pepper, alcohol.

(b) In peritonitis, intestinal obstruction, and many acute abdominal diseases.

(c) Uraemia. The cause of the hiccough in this condition is not clear.

(d) In certain lesions of the nervous system. Epidemics of hiccough are described, usually during an epidemic of encephalitis, but the two conditions do not necessarily coexist. Sometimes hiccough is a feature of acute intracranial disease. Hysterical hiccough may occur.

The treatment of the mild type is to hold the breath, or to relieve the flatulence. Many tricks are practised by patients to abolish the symptom. In the severe forms many treatments have been tried without constant effect. It may be sufficient for the patient to bite some solid object, such as a cork, or bromide may be given, but in obstinate cases morphia or inhalations of chloroform may be necessary.

*Diaphragmatic pleurisy.*—In addition to the general features of acute pleurisy there are certain special symptoms when the diaphragm is involved. Pain is likely to be referred to the shoulder or to the abdomen; local signs will be slight, or even absent. The diagnosis is therefore often difficult.

There may be tenderness and rigidity of the abdominal muscles and the clinical appearance may closely resemble that of acute abdominal disease, such as perforated ulcer or acute appendicitis. Whenever the signs of an "acute abdomen" are in any way atypical, it is necessary carefully to consider the possibility of diaphragmatic pleurisy before embarking on laparotomy.

*Epidemic diaphragmatic pleurodynia.*—This is otherwise known as "Bornholm disease" or "devil's gripe". It appears to be an acute inflammation of the muscle itself and of the adjacent pleura. Children and young adults are chiefly affected, and the disease is transmitted by contact, occurring in epidemics. It is clearly an infection, most likely by a virus.

The onset is sudden, with a chill or rigor, and a rise of temperature to about  $103^{\circ}$ . There are the constitutional disturbances of an acute infection, and sweating is common. The chief symptom is acute pain, which begins in the epigastrium and settles later in either side of the chest. It is worse on inspiration, so that the respirations are usually



shallow, and it may be referred to the shoulder. There is no cough nor sputum.

There is local tenderness of the muscles and, in about half the cases, pleural friction. The white count is often normal but it may be somewhat raised, the main increase being in the polymorphs.

The pain comes on in paroxysms and lasts for two or three days, but there is a tendency to relapse. Treatment is entirely symptomatic, by means of aspirin and the local application of heat, and recovery is always complete.

# INDEX

- Abcess of lung, 197
  - X-ray appearances, 69
- Accessory lobe, 68
- Actinomyces, 72, 83, 257
- Added sounds, 47
- Adenoids, 93
- Adhesions, division of, 218
- production, 269
- Agrophony, 49
- Air embolism, 158, 249
- Air replacement, 281
- Alar chest, 32
- Allergy in asthma, 117
- in tuberculosis, 216
- Anemorrhoea, 24, 216
- Amphoric breathing, 47
- Amyloid disease, 132
- Anaerobes, 82
- Aneurysm, 294
- X-ray appearances, 57
- Anthrax, 263
- Apex beat, 39
- Aphonia, hysterical, 21
- Arthritis in pulmonary tuberculosis, 25
- Artificial pneumothorax, 242
- Asbestosis, 263
- Aspiration of chest, 280
- pneumonia, 188
- Asmann's focus, 218
- Asthma, spasmodic, 117
- investigation of, 121
- treatment of, 123
- Asymmetry of the chest, 33
- Atelectasis, 69, 131, 165
- Atelectatic crepitations, 48
- Atypical pneumonia, 193
- Auscultation, 43
- Axillae, palpation of, 38
- Ayerza's syndrome, 18, 152
- Azygos vein lobe, 68
- Bacillus pertussis*, 81
- Bacillus tuberculosis*, 62
- Balamoia, 264
- B.C.G. vaccine, 211
- Benadryl, 95
- Black spit, 7, 73, 263
- Bornholm disease, 304
- Drain abscess, 132
- metastases, 141
- Breath sounds, 45
- Breathing exercises, 126
- Brompin, 63
- Bronchial breathing, 46
- Bronchial carcinoma, 61, 134
- Bronchial obstruction, 144, 160, 267
- Bronchiectasis, 130
- clinical types, 133
- cough in, 2
- haemorrhagic, 133
- treatment, 136
- Bronchitis, acute, 109
- chronic, 113
- fibrinous, 7
- Bronchogram, 63, 135
- Bronchophony, 49
- Broncho-pleural fistula, 287
- lipiodol appearances, 68
- Bronchopneumonia, 187
- tuberculous, 216
- Bronchorrhoea serosa, 7, 72
- Bronchoscopy, 82, 144
- "Broncho-squamous disease", 2, 69
- Broncho-vascular breath sounds, 47
- Bronchus, carcinoma of, 61, 138
- foreign body in, 109
- innocent tumour of, 147
- obstruction, 144, 160, 267
- syphilitic, 116
- tuberculosis of, 116
- Bullos in emphysema, 150
- Dyspnoea, 264
- Cachexia, 30
- Calcification, 60, 207
- Campbell de Morgan's spots, 24, 36
- Carbon dioxide, therapeutic uses, 169
- Carcinoma of bronchus, 138
- X-ray appearances, 61
- Cardiac impulse, 35
- Caution of tuberculosis, 225
- Catarrh, 208
- Cavity, physical signs, 224
- X-ray appearances, 69
- Cerebral abscess, 133
- metastases, 141
- Charcot-Leyden crystals, 74, 122
- Cheek-valve obstruction, 163
- Chemotherapy, 183
- Chylothorax, 84, 274, 279
- Clubbing of the fingers, 37

- Coarctation of the aorta, 54  
 Cocaine sensitiveness, 63  
 Cog-wheel breathing, 46  
 Coin sound, 50  
 Collapse of lung, 59, 164  
 Collapse treatment of tuberculosis, 242  
 Complementary air, 16  
 Congenital laryngeal stridor, 105  
 Congestion of the lungs, 153  
 Consolidation of lung, 40, 43, 47, 60, 176  
 Coryza, 95  
 Cough, causes of, 1  
     "brassy", 4  
     change of position, 3  
     due to habit, 5  
     due to pressure, 4  
     reflex, 11  
     remote results from, 5  
 Cracked-pot sound, 43  
 Crepitations, 48  
 Crisis in pneumonia, 177  
 Croup, 107  
 Cup-shaped hollow, 33  
 Curschmann's spirals, 74, 122  
 Cyanosis, 30  
 Cyrtometer, 31  
 Cyst, bronchiectatic, 130  
     dermoid, 293  
     hydatid, 61  
 Delayed resolution, 177  
 Dermoid cyst, 293  
     X-ray appearances, 57  
 Devil's gripe, 301  
 Diaphragm, eventration of, 55, 300  
     paradoxical movement, 52, 300  
     paralysis of, 55, 300  
     X-ray appearances, 55  
 Diaphragmatic hernia, 55, 299  
     pleurisy, 13, 301  
     pleurodynia, 301  
 Diphtheria, laryngeal, 106  
 Dittrich's plugs, 73  
 Double-exposure X-ray films, 69  
 Dumb-bell tumour, 294  
 Dyspepsia in respiratory disease, 23  
 Dysphagia, causes of, 23  
 Dyspnoea, 16  
     in heart-failure, 18  
     in pneumonia, 175  
 Effusion, pleural (*see* Pleural effusion)  
 Elastic fibres in sputum, 74  
 Embolism, air, 158  
     fat, 158  
     pulmonary, 155  
 Emphysema, acute vesicular, 162  
     atrophic, 162  
     compensatory, 162  
     hypertrophic, 159  
     interstitial, 163  
     localized, 163  
     "surgical", 39, 48, 163  
     X-ray appearances, 58  
 Empyema, 283  
     chronic, 286  
     metapneumonic, 284  
     necrotic, 286  
     synpneumonic, 284  
     tuberculous, 287  
 Endothelioma of pleura, 280  
 Eosinophil cells in sputum, 75  
 Epituberculosis, 214  
 Erythema nodosum, 24, 27  
 Erythrocyte sedimentation rate, 86, 227  
 Eventration of diaphragm, 55, 300  
 Expansion of chest, 34  
 Expiration, prolongation of, 46  
 Extra pleural pneumothorax, 252  
 Exudate in pleura, 87, 274  
 Family history, 23  
 Fat embolism, 158  
 Fatigue, 22  
 Fibrosis of lung, 259  
     X-ray appearances, 60  
 Fistula, broncho-pleural, 68, 287  
 Fistula-in-ano, 24, 231  
 Flat chest, 32  
 Fluid level, 53, 59  
 Foreign body, in bronchus, 3, 109  
     in larynx, 101  
     in lung, 150  
 Foulis' cells, 87, 145  
 Friction, pleural, 48  
 Friedlander's bacillus, 81  
 Fungus infections, 257  
 Fusiform bacilli, 84  
 Gangrene of the lung, 197  
 Gee's linctus, 116  
 Ghon's focus, 213  
 Glands in hilum, 56, 291  
 Glegg's mixture, 95  
 Glottis, oedema of, 102  
 Goitre, retrosternal, 57, 293  
 Gold treatment, 241  
 Grocco's triangle, 275  
 Habit cough, 5  
 Haemopneumothorax, 270

- Haemoptysis, causes of, 8
- investigation of, 10
- recognition of, 8
- treatment of, 253
- vicarious, 10
- Haemothorax, 278
- Harrison's sulcus, 33
- Hay fever, 98
- Heart, displacement of, 39
- Heart failure cells, 75
- Hematite miner's lung, 264
- Hepatization, 174
- Hereditary factors in chest disease, 28
- Hernia of the diaphragm, 55, 299
- of lung, 150
- Herpes, labial, in pneumonia, 175
- zoster, 14
- Hiccough, 309
- Hilar glands, 86, 201
- History-taking, 1
- Honeycomb lung, 130
- Homer's syndrome, 143, 147
- Hydatid cyst, 61
- hooklets in sputum, 74
- Hydropneumothorax, 268
- Hyperpnoea, 16
- Hypertrophic pulmonary osteoarthro-  
pathy, 23, 132
- Hypostatic pneumonia, 154
- Hysterical dyspnoea, 18
- Incus crepitations, 48, 175
- Infarct of lung, 156
- Infiltration, 60
- Influenza, 191
- Injury to the chest, 149
- and tuberculosis, 26, 151, 210
- Inspection, general considerations, 29
- Intestinal tuberculosis, 229
- treatment, 264
- Iodine sensitiveness, 63
- Joint conditions in respiratory disease,  
23
- Kopp's asthma, 107, 294
- Kymograph, 69
- Kyphosis, 33, 54
- Laryngismus stridulus, 106
- Laryngitis, acute, 101, 106
- chronic, 102
- stridulous, 106
- sphyilitic, 105
- tuberculous, 103
- Larynx, foreign body in, 101
- oedema of, 102
- Larynx—(contd.)
- papilloma of, 107
- Lipiodol in bronchial carcinoma, 144
- in bronchiectasis, 135
- indications for, 67
- residual, in lungs, 67
- technique of, 63
- Lipoid pneumonia, 193
- Litten's sign, 37, 300
- Louis' law, 210
- Lowenstein's medium, 83
- Lung abscess, 197
- Lung stone, 207
- Lung tumours, 148
- Lymphadenoma, 292
- Lymphosarcoma, 293
- Lysis, 177
- Malaise, 22
- Mantoux test, 210
- Marital tuberculosis, 208
- Mass radiography, 69
- Massive collapse of lung, 166
- Massive necrosis of lung, 107
- Mediastinal glands, 291
- Mediastinitis, 290
- Mediastinum, displacement of, 35, 39,  
66
- tumours of, 140, 292
- X ray appearance, 56
- Meningismus, 180
- Meningitis, pneumococcal, 180
- tuberculous, 217
- Metallic tinkle, 50
- Metapneumonic empyema, 244
- Metastases in lungs, 61, 143
- Micrococcus catarrhalis, 81
- Miliary tuberculosis, 207, 216
- Monaldi drainage, 251
- Movement of the chest, 34
- Myoidema, 38
- Myotatic irritability, 38
- Nasal factor in asthma, 118
- Nasal passages, 91
- Nervous system, changes in chest  
disease, 25
- Night sweats, 22
- Nummular sputum, 73
- Oat celled carcinoma, 139
- Occupation and chest disease, 4, 26
- Oedema of glottis, 102
- of lung, 154
- Oleothorax, 249
- Opalescent pleural fluid, 84, 274
- Orthopnoea, 19
- Oxygen administration, 186

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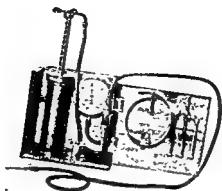


FIG 1.—The author's pneumothorax apparatus

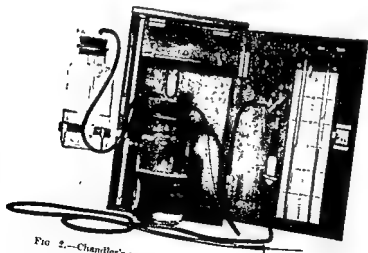


FIG 2.—Chandler's modification of the Lillingston-Pearson pneumothorax apparatus

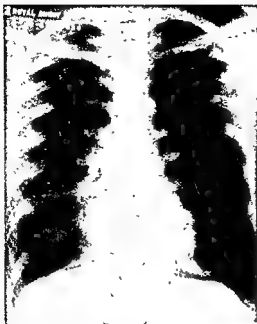


FIG. 3.—Normal chest



FIG. 4.—Scoliosis



FIG. 5.—Accessory lobe of the axylar vein: the vertebral borders of the scapulae are seen



FIG. 6.—Eventration of the diaphragm





FIG. 7.—Normal right bronchogram, anterior view



FIG. 8.—Normal right bronchogram, lateral view

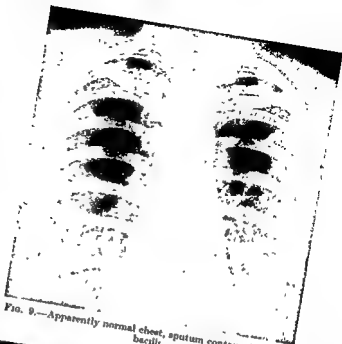


FIG. 9.—Apparently normal chest, sputum contained tubercle bacilli

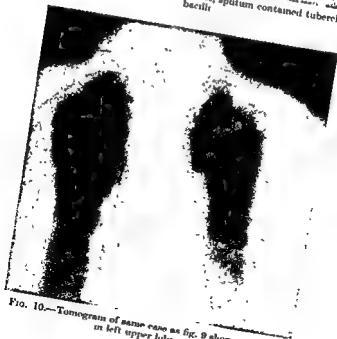


FIG. 10.—Tomogram of same case as fig. 9 showing cavity in left upper lobe



FIG. 11 — Large right-sided pleural effusion



FIG. 12.—Right-sided artificial pneumothorax



FIG 13.—Massive collapse of the left lung, carcinoma of the left main bronchus



FIG 14.—Dense fibrosis of right lung, chronic tuberculosis



FIG. 15.—Congenital cystic disease, right upper lobe



FIG. 16.—Congenital cystic disease, right upper lobe, bronchogram taken with patient supine

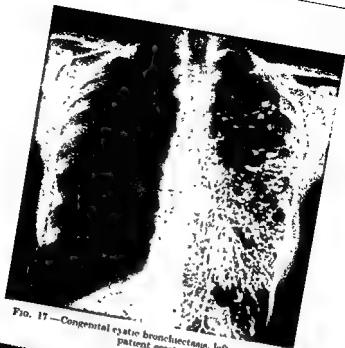


FIG. 17—Congenital cystic bronchiectasis, left upper lobe, patient erect

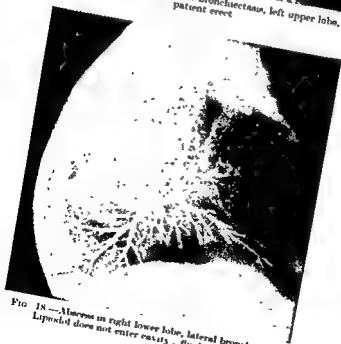


FIG. 18—Abscess in right lower lobe, lateral bronchogram  
Lipiodol does not enter cavity. fluid level visible



FIG. 19.—Acquired bronchiectasis, left lung



FIG. 20.—Bronchiectasis in right lower lobe, lateral view

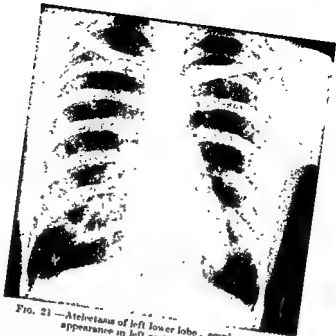


FIG. 21.—Atelectasis of left lower lobe, emphysematous appearance in left costophrenic sinus

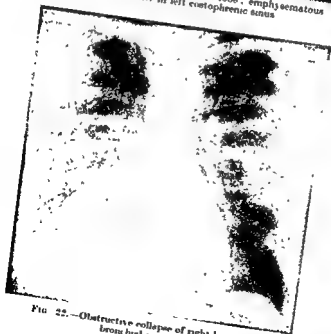


FIG. 22.—Obstructive collapse of right lower lobe bronchial carcinoma





FIG. 23.—Lung abscess showing fluid level (see fig. 24)



FIG. 24 —Lung abscess in right middle lobe (see fig. 23);  
lateral view

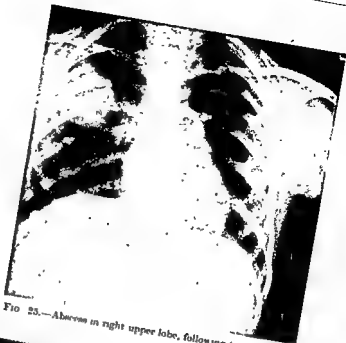


FIG 25.—Abscess in right upper lobe, following tonsillectomy

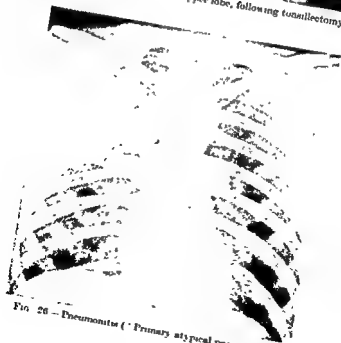


FIG 26 — Pneumonitis ("Primary atypical pneumonia")



FIG. 27.—Bronchial carcinoma, tumour in right hilum



FIG. 28.—Bronchial carcinoma, tumour in right upper lobe



FIG. 29.—[Bronchial carcinoma simulating mediastinal tumour]



FIG. 30.—[Bronchial carcinoma in right lung  
paralysis of the right diaphragm]



FIG. 31.—Bronchogram, left main bronchus obstructed by innocent tumor



FIG. 32 —Left lateral view, bronchial block due to carcinoma



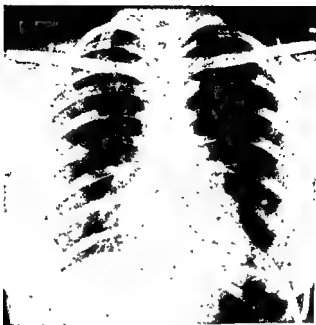


FIG. 35.—Calcified Ghon's focus, left mid zone, and gland in hilum

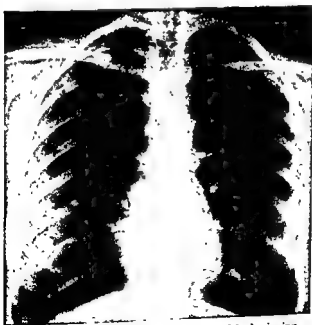


FIG. 36.—Assmann's focus below left clavicle, beginning to break down



FIG 37.—Tuberculous bronchopneumonia

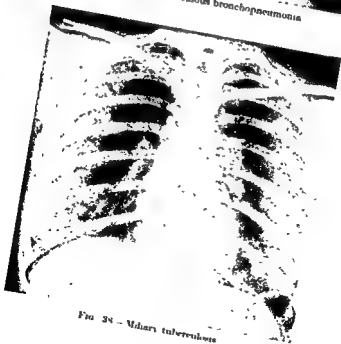


FIG 38 — Miliary tuberculosis



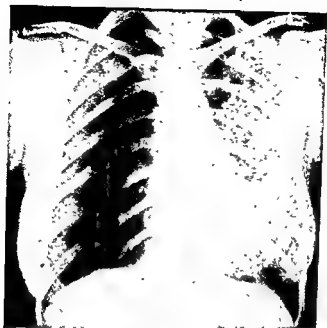


FIG. 39.—Tuberculous infiltration



FIG. 40—Residual lipiodol in right lung

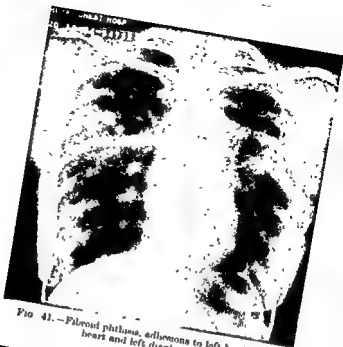


FIG. 41. — Fibroid phthisis, adhesions to left border of heart and left diaphragm

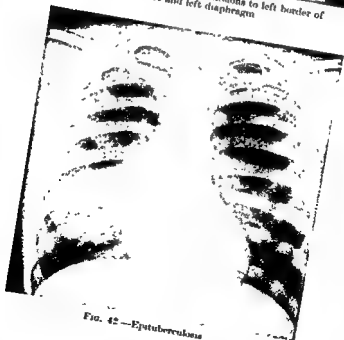


FIG. 42. — Epituberculosis



FIG. 43.—Large tuberculous cavity at right apex with some surrounding infiltration



FIG. 44.—Thoracoplasty for treatment of pulmonary tuberculosis; calcified primary focus in right lung



FIG 45—Silicosis

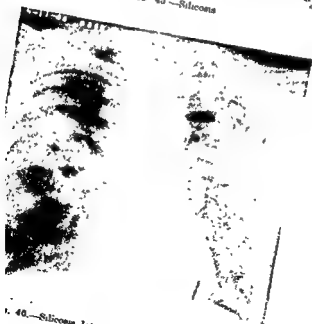


FIG 46.—Silicosis, later stage with nodule formation.  
atheroma of the aorta



FIG. 47.—Small pleural effusion



FIG. 48.—Oleothorax compressing cavity in left upper lobe



FIG. 49 — Right sided hydro-pneumothorax



FIG. 50 — Left sided artificial pneumothorax. apex held up by breast adhesion



FIG. 51.—Broncho-pleural fistula; lipiodol injected through a sinus has entered the bronchi in both lungs



FIG. 52.—Dermoid cyst in right anterior mediastinum

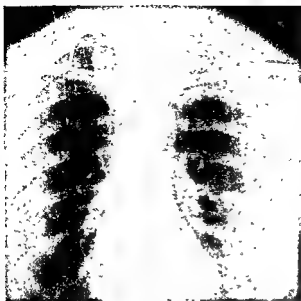


FIG. 53 — Retrosternal goitre showing displacement and compression of the trachea



FIG. 54.—Lymphadenoma, masses of enlarged glands in the mediastinum





FIG. 55.—Fusiform aneurysm of the aorta



FIG. 56.—Saccular aneurysm of the aorta



FIG. 57 —Lymphosarcoma in the mediastinum (see fig 54)

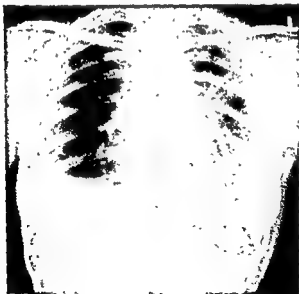


FIG. 58 —Same case as fig 57 after three weeks' deep X ray treatment

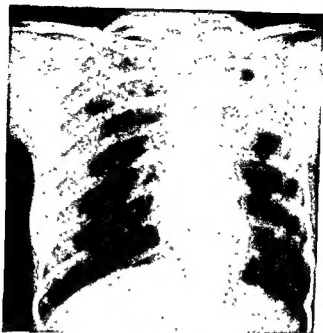


FIG. 59.—Pulmonary tuberculosis, large cavity in left upper lobe, and infiltration in right upper lobe

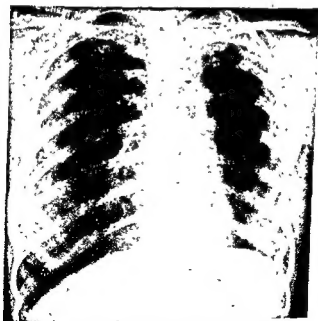


FIG. 60.—Same case as Fig. 59 after phrenic avulsion, left diaphragm is raised and the cavity has disappeared

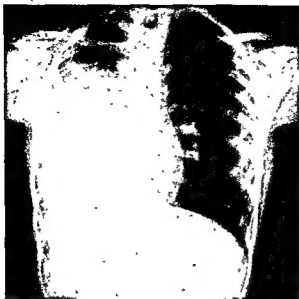


FIG. 61.—Hernia through the right diaphragm



FIG. 62 — Same case as fig. 61. Shows the hepatic flexure of the colon.



FIG. 63.—Normal bronchial mucous membrane

FIG. 64.—Columnar-celled carcinoma

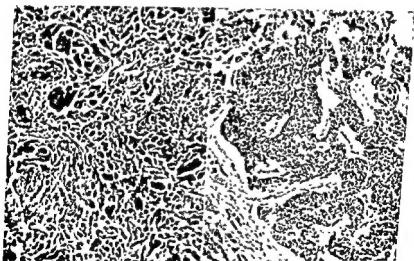


FIG. 65.—Squamous-celled carcinoma

FIG. 66.—“Oat”-celled carcinoma

